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LINES OF GROWTH IN LONG BONES OF INDIANS

A Preliminary Report

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Transverse striations are occasionally seen in skiagrams of long bones. In view of their incidence at particular periods of life and their association with the growth cartilage they are classified as follows:—

1. Those appearing at the site of epiphysial cartilage after union and cessation of growth; hence recognised as C. G. lines.
2. Those appearing at the metaphysis earlier and attributed to arrest of growth; hence recognised as A.G. lines (Figs. 1-4).

Our attention was drawn to so-called A.G. lines recently in the course of an investigation on epiphysial union in girls of U.P. As the subject has not received any attention so far in this country it was considered desirable to study their incidence and significance.

Material and Methods

Human as well as animal skeletons have been studied. Radiological and histological observations on the growing ends of long bones have been carried out.

Clinical history in case of human subjects has been correlated with the radiographic and histological findings in the bones. We could secure 17 positive radiographs out of 73 examined. Findings have been summarised in a tabular form.

Historical

Transverse striations other than C.G. lines of long bones were first observed by workers on rickets *viz.*, Wegner (1874), Kassowitz (1884), in the course of their experimental work on rabbits fed with minute quantities of phosphorus. Several anatomists, surgeons and radiologists contributed some material in the form of case reports between 1903 and 1926.

The significance of these lines was studied by various workers Clinical and experimental observations prompted these workers to formulate a variety of hypothesis regarding their genesis: one view attributed them to different rates of ossification at different ages; while another worker regarded them as representing a period in which better bone was laid down as a result of improvement in the general condition; Howland and Park's discovery (1921) of the 'line tests' for healing rickets captured the imagination of workers in the field for some time and every transverse striation was explained in terms of healing rickets.

Recent workers regard it as evidence of arrest of growth due to grave metabolic disturbance associated with disease. During the whole period of growth, it is maintained, any severe interference with metabolism, either as a result of starvation, acute infection or metabolic disease, such as diabetes, induces premature senescence in growing tissues and the fact is unfailingly registered in the structure of the bone by the production of a transverse line of arrested growth. Such views are contended by others who consider that a generalization of this nature is fallacious.

Material for Study

Altogether 17 cases were studied, ages ranged from 5 to 50 years. They generally came for x-ray of a part of the body due to other conditions, *e.g.*, fracture, synovitis etc. The incidence, clinical history and site of A.G. lines are summarised in Table I.

Perusal of this summary gives some idea regarding incidence of A. G. lines Striations in this small series are confined to knee, ankle and wrist; they tend to be asymmetrical. Clinical history does not offer any help in majority of the recorded cases. Their varying number and distance from the metaphysis is suggestive of a variety of causes at different periods of life.

An extensive clinical and experimental study is required to assess their significance. Observations made by us and previous workers warrant the following conclusions:

(*i*) That both A. G. and C. G. lines enjoy a common parentage—the growth cartilage.

(*ii*) That both present an identical structure.

(*iii*) That reference to the biography of the growth cartilage in relation to the genesis of C. G. lines is helpful in appreciating the mechanism of production of A. G. lines.

Origin—It has descended from the cartilaginous blastema of the limb bud and apears to have been trapped between epiphysis and diaphysis to contribute to their post-natal growth. Cicumferential extensions from this disc of embryonic cells underneath the articular cartilage constitute the mitotic annulus (Fig. 5). While the disc enjoys a limited span of life its articular extension copes throughout with wear and tear at the joint surface.

Structure—Our conception is based upon its classical description by Broca and Ranvier supplemented by personal observations by one of us on human

TABLE I—SHOWING INCIDENCE, CLINICAL HISTORY AND SITE OF A.G. LINES

Sl. No.	Plate No. & Year	Site	Age	Sex	Condition for which skiagraphed	Past history	No. & distance of A.G. lines from metaphysis
1	82/41	Wrist (rt.)	22	M	Fracture, radius (distal)	Jaundice, 6 yrs.	Radius—one line; 9 m.m.
2	165/41	Rt. tibia (distal)	40	M	Fracture tibia & fibula	..	One line; 10 m.m.
3	202/41	Rt. tibia (distal)	13½	M	Painful heel	T.B. adenitis (?)	One line; 15 m.m.
4	589/41	Rt. tibia (distal)	9	F	T.B. rt. foot	Small pox, 1 yr.	Two lines; 10 & 18 m.m.
5	382/42	Rt. tibia (distal)	6	M	Fracture tibia	Small pox, 3 yrs.	One line; 13 m.m.
6	936/42	Rt. knee joint	15	M	Ch. synovitis lt. knee	Intermittent fever for 1 yr.—3 yrs. (back)	Femur—3 lines; 15, 25 & 35 m.m. Tibia—2 lines; 7 & 10 m.m.
7	989/42	Knee joint rt. & lt.	11	F	Pain in lt. T.B. knee lt. elbow—7 months	High fever twice (date not known)	Femur—4 lines; Tibia—4 lines; Fibula—3 lines—within 10 m.m.
8	1574/42	Lt. knee	6	F	T.B. knee	..	Femur—3 lines; 2, 5 & 18 m.m. Tibia—4 lines; 3, 7, 10 & 15 m.m.
9	1753/42	Rt. tibia (distal)	23	M	Sprain ankle	Malaria, 2 months Dysentery, 1 month	Tibia—3 lines; 17, 30 & 43 m.m.
10	79/43	Femur (distal)	14	M	Fracture femur	..	Single line; 34 m.m.
11	102/43	Lt. ankle	50	M	Path. fracture with osteomyelitis	..	Tibia epiphysial scar & 2 lines; 9, 20 m.m.
12	463/43	Rt. femur (distal)	12	M	Osteomyelitis femur	..	Single line; 14 m.m.
13	1128/43	Rt. wrist	12	M	Fracture radius & ulna	..	Radius—2 lines; 4 & 11 m.m.
14	1504/43	Lt. ankle	11	M	Osteomyelitis tibia	Pneumonia—3 yrs. Occasional fever	Tibia—single line; 4 m.m.
15	1767/43	Rt. ankle	20	M	Osteomyelitis rt. tibia upper ¾	Osteomyelitis, 7 years.	Tibia—single line; 17 m.m.
16	1803/43	Lt. knee & ankle	5	M	Osteomyelitis tibia upper ¾	..	Femur—3 lines; 6, 12 & 27 m.m. Tibia (distal end)—2 lines; 5 & 8 m.m. Fibula—out of focus.
17	Special	Rt. knee	16	F	X-rayed for age determination	Cold & fever off and on.	Femur—2 lines; 10 & 23 m.m. Tibia—2 lines; 10 & 18 m.m.

as well as animal material. Histologically it comprises of three distinction zones (Figs. 7 and 8):

(i) *Couche chondroide* (C_1) comprises of translucent columns of cells with large nuclei, intercolumnar matrix, pronounced at sites of active growth (knee, ankle and wrist).

(ii) *Couche osteoide* (C_2) presents an opaque granular appearance due to calcareous deposits around cartilage cells in longitudinal and transverse axes, giving rise to longitudinal and transverse trabeculæ respectively. These cells are discrete, large, rectangular and contain small nuclei. Cartilage cells. thus imprisoned, start undergoing degeneration and disappear completely by the time the calcified reticulum is transformed into an organised three-directional lattice-work

(iii) *Couche osseuse* (C_3) presents the process of ossification in a previously calcified band. Its outstanding feature is invasion by medullary and periosteal vessels accompanied by osteoblasts and osteoclasts resulting in the demolition of most of the transverse and some of the longitudinal trabeculæ. Those that survive constitute the spongy structure of bone wherein longitudinal trabeculæ predominate throughout the period of active growth.

Nourishment—It is derived from (*i*) 'circulus vasculosus' of Hunter and (*ii*) virtual end-arteries of the diaphysis. There is hardly any anastomosis between the two sets of vessels (Fig. 6).

The nutrition of each cell is effected by imbibition of tissue juice, rich in water-soluble, growth-stimulating products, which diffuse through the matrix. Each cell grows to attain a maximum size and divides into the maximum number of cells, which can thus be nourished. The peak, thus reached, predisposes individual cells to undergo senescence and degeneration while the matrix around them as well as in between their columns is calcified. If such be the fate of these cells under optimum conditions what will their response to poverty of nourishment be? Perversion of behaviour is the obvious reply; its actual nature will vary with the degree and type of deficiency.

Fate—Its ultimate fate is ossification *en bloc*. This is effected by a preliminary failure of mitosis in the first, intense calcification in the second and heavy ossification in the third zone. Following this the first zone is calcified while ossification extends into the second. Thus growth ceases for ever. Finally the entire disc is ossified and results in fusion of an epiphysis with its diaphysis; structural homogeneity

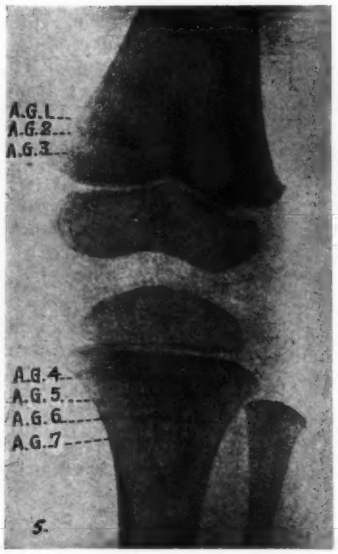


FIG. 1—RADIOGRAPH OF THE LEFT KNEE OF A FEMALE AGED 11 YEARS. TIBIAL PATTERN ASYMMETRICAL

FIG. 2—RADIOGRAPH OF LEFT ANKLE OF A MALE AGED 13½ YEARS. PATTERN SYMMETRICAL

FIG. 3—RADIOGRAPH OF THE TIBIA OF A GIRL AGED 16 YEARS

FIG. 4—RADIOGRAPH OF THE LEFT WRIST OF A MALE AGED 20 YEARS. PATTERN ASYMMETRICAL

FIG. 7—MICROPHOTOGRAPH OF THE FEMUR OF A HUMAN FOETUS SHOWING STRUCTURE OF GROWTH CARTILAGE

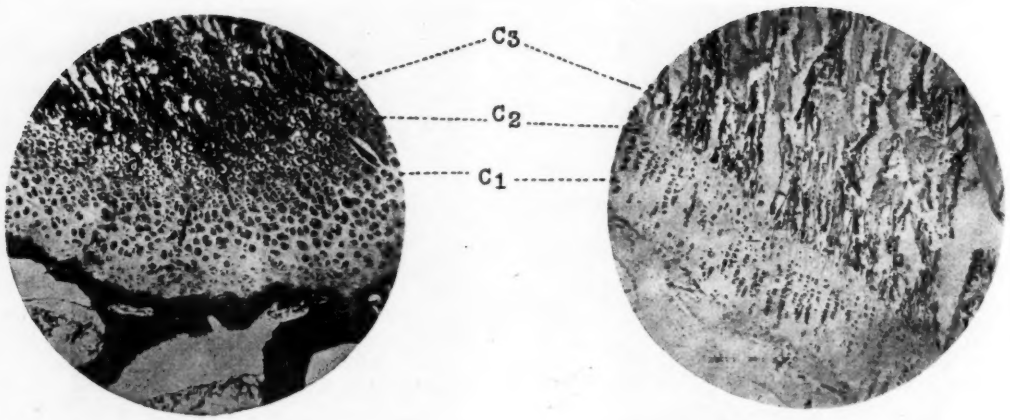
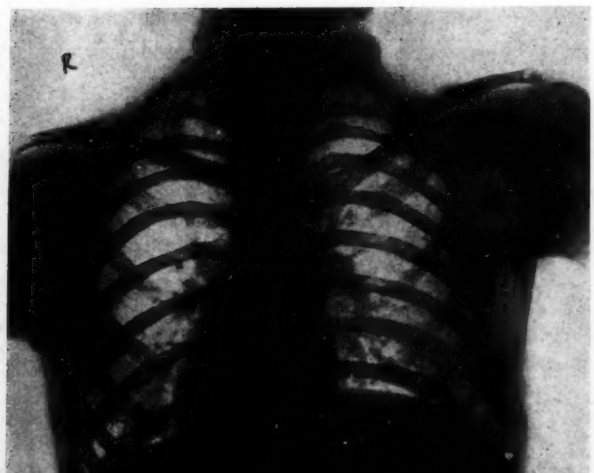


FIG. 8—MICROPHOTOGRAPH OF THE FEMUR OF A RABBIT SHOWING STRUCTURE OF GROWTH CARTILAGE

KHAN & CHATTERJEE—*Lines of Growth in Long Bones of Indians*



SKIAGRAM SHOWING DEXTROCARDIA

RAO—*Dextrocardia*

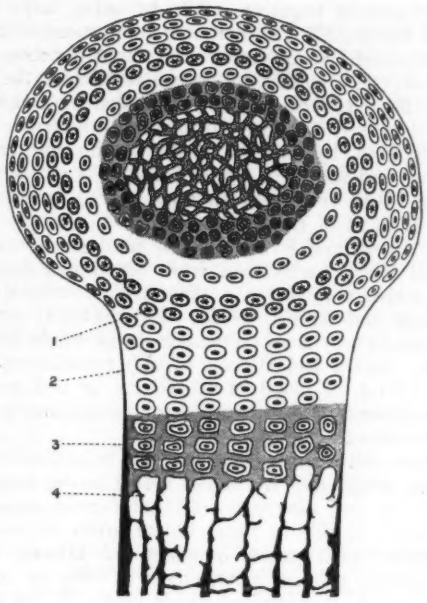


FIG. 5—DIAGRAMMATIC MITOTIC ANNULUS (AFTER HARRIS)
1. Mitotic annulus
2. Couche chondroide
3. Couche osteoide
4. Couche osseuse

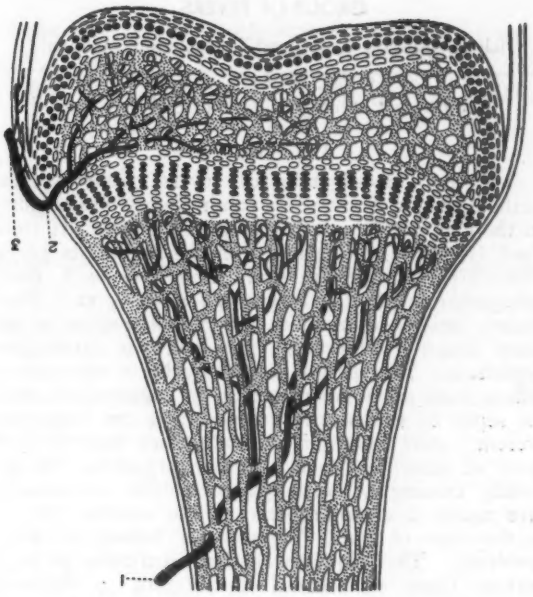


FIG. 6—DIAGRAM TO SHOW THE BLOOD SUPPLY OF THE GROWTH CARTILAGE (AFTER HARRIS)
1. Diaphysial artery
2. Epiphysial artery
3. Circulous vasculosus

across its site is soon established. The entire process may extend over 6-24 months or even longer and presents a graded picture of radiological union.

Genesis of C. G. Lines—One peculiarity of its terminal ossification merits special mention, *viz.*, failure of absorption of transverse trabeculæ. Consequently, they are allowed to retain their original dimensions. Predominance of these 'transoms' persists even after fusion and according to an author "accounts for the constancy with which the site of the growth cartilage is marked in adult bones by a line of completely arrested growth in which the transverse trabeculæ are more heavily developed than the longitudinal trabeculae". This line in its turn accounts for the transverse striation in radiographs of adolescent bones at the site of the epiphysial cartilage. It tends to disappear as a result of remodelling in due course, but may persist in part or wholesale in rare cases long after bony union and accounts for the 'epiphysial scar' of radiologists. Predominance of transverse trabeculæ, in like manner, is believed to characterise its ossification during arrest of growth in earlier life and explains the radiographic occurrence of A. G. lines. Hence the mechanism of production of A. G. and C. G. lines are identical.

Significance of C. G. line—What factors are responsible for cessation of growth in the epiphysial cartilage? Cessation is essentially an age-phenomenon and hinges on the state of metabolic and endocrine equilibrium; other factors deserving mention are heredity, sex, race, environments etc. The first part of the remark needs further explanation. Mitosis at each growth cartilage is allowed to attain a peak in respect of both the size and number of cells in its component columns. Climax of mitotic activity is followed by senescence and degeneration of cells; calcification and ossification in their matrix after a certain pattern under the stimulus of the attainment of adult stature. This view finds support in the retardation of epiphysial union being associated with hypo and acceleration with hyperactivity of certain endocrines, *viz.*, thyroid, pituitary, gonads etc.

Disturbance of metabolic equilibrium in early life manifests itself in a variety of ways: (*i*) Impaired rate of growth, a border line between health and disease, (*ii*) Disease, rickets and scurvy, (*iii*) Arrest of growth, a mechanism of defence to tide over an emergency. Further details about the last-mentioned phenomenon and its relation to metaphysial striations will form the basis of a subsequent communication.

OBSERVATIONS ON BLOOD CULTURE IN ENTERIC GROUP OF FEVERS

(Using Gentian Violet and Taurocholate Medium)

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INTRODUCTION

In the investigation of fevers, bacteriological methods are in recent years gaining much prominence. In the case of the enteric group of fevers, apart from *Bact. typhosum,* the members of the salmonella group which are causative, are *Bact. paratyphosum* A, *Bact. paratyphosum* B, *Bact. paratyphosum* C, and *Bact. cholera* sui-var-kunzendorf. *Bact. suipestifer* is the name found in literature for both the latter two organisms. The disease caused by these two species differs from other enteric group of organisms in that the septic or suppurative complications are frequently present. *Bact. enteritidis* has also been isolated from cases of continued fever. Other organisms though usually causing an acute gastroenteritis, occasionally give rise to a continued fever of the enteric type, as in the case of *Bact. typhimurium* known as *Bact. aertrycke.* The frequency of the distribution of these various types of bacteria is different in different countries, and this fact is of great significance from the point of view of diagnosis.

The direct proof of a person suffering from enteric fever is the isolation of *Bact. typhasum* or the allied organisms from his blood, urine or stool, and the demonstration of the presence of specific agglutinins affords indirect evidence. Topley and Wilson emphasising that the actual isolation and identification of the infecting organisms is always the method of choice, indicated that a positive agglutination reaction may be highly suggestive and in some cases practically conclusive but it should not be accepted as satisfactory evidence of enteric infection except in those rare cases in which bacteriological examination of the blood and excreta is impossible. The organisms of enteric fever can be isolated in the blood in practically every case of enteric fever in the first week of the disease, in about 80-85 per cent of cases in the second week and in decreasing percentage in later weeks. Where the laboratory worker has no access to the patient a clotted blood sent for an agglutination test can better be used for the isolation of enteric organism. In an early case where the agglutination test remains negative clot culture forms a very helpful means for the early and current diagnosis (Soman 1932, 1934 and Downie and Fairbrother 1934). The present paper deals with the observations on 56 cases of enteric fever in Agra using the new gentian violet and taurocholate medium for blood culture. The efficacy of this medium as compared with the other media is also discussed.

TECHNIQUE

I. Media—Castellani (1899-1902) and Schottmular (1902) were first to report results indicating the true frequency of blood stream invasion in the disease. Castellani attempted cultivation in broth and showed that with small amounts of media the blood cultures were frequently negative, while by using large quantities of broth (300 c.c. or more) many positive results were obtained. Schottmuller considered it necessary to use 10-20 c.c. of blood. The use of bile for the isolation of *Bact. typhosum* was introduced by Conradi in 1906. He used a mixture of bile and glycerine. This was modified by Kayser (1906) who omitted the glycerine and used 2·5 c.c. of blood to 5 c.c. of sterile ox bile. Bile or bile broth now has so far been the choice of most of the workers.

Le Boeuf and Braun (1916) used sterile urine in place of bile. Cultivation from blood clot has been found satisfactory for the diagnosis of enteric fever (le Boeuf and Braun, 1906). Felix (1924) found it more satisfactory than cultivation from whole blood in ox bile. Topley and Wilson (1929) recommended the use of 10 c.c. of blood in 50-100 c.c. of 0·5 per cent sodium taurocholate in broth or peptone water or even in water alone.

Plate culture has been used by Schottmuller as a guide to prognosis. A set of media to be used as a routine in the diagnosis of bacteraemic conditions consists of: 1. Bile or taurocholate broth or taurocholate gentian violet alone in water. 2. Glucose broth. 3. Anaerobic cooked meat medium with or without trypsin and 4. Trypsinised digest broth. If the patient is not suffering from enteric fever but from some other bacteraemic infection, the use of media which do not contain bile or bile salts frequently enables the causal organism to be cultivated from the blood.

Clot culture particularly requires the use of media inhibiting staphylococci, as the required aseptic methods are usually not followed in taking blood for agglutination and the skin contaminants are often grown from the clot. Gentian violet taurocholate inhibits staphylococci and permits the growth of a small number of typhoid bacilli. A simple broth medium containing 0·1 per cent sodium desoxycholate adjusted to pH 7 was found to be highly successful by Cruickshank (1943).

I. Technique used—(*i*) Gentian violet 1/500,000 in nutrient agar is known to inhibit the growth of staphylococci and this has been used with 2 per cent sodium taurocholate solution in distilled water for the isolation of enteric group of organisms. It should be of much benefit when using clot culture for the diagnosis of enteric fever. 1 c.c. of 1/2,500 gentian violet is added to 100 c.c. of 2 per cent. taurocholate solution in distilled water and this is distributed in 20 c.c. amount in sterile test tube and the media is sterilised in an autoclave.

(*ii*) All-glass 20 or 10 c.c. syringe sterilised in a hot steriliser and wide bore needles of size 30/1 mm. have been used. Small and fine bore needles are to be avoided.

(*iii*) The skin of the patient's forearm is cleaned with spirit and other.

(*iv*) 15 c.c. of blood is aseptically withdrawn from the patient's forearm and 5 c.c. of this is added to the gentian violet taurocholate medium and the rest is distributed to the anaerobic and glucose broth and

trypsin digest broth media. The media are inoculated by the side of the patient's bed.

(*v*) Cultures are placed in the incubator as soon as possible after the blood is added and at the end of twelve and twenty-four hours are examined for motile bacilli by the hanging drop method. If none is found transfers are made to slants of nutrient agar and incubated for twelve or twenty-four hours and if negative, for a longer time. If motile gram-negative bacilli are found they are presumably typhoid or paratyphoid bacilli. The agglutination test has been used with specific high titre sera. When this is negative the biochemical reactions are studied by inoculating glucose, lactose, mannite and maltose sugar media. When these are inconclusive further tests are done. The primary cultures are kept in the incubator for as long as two to three weeks.

Results

During the year 1945, 56 cases of enteric fever were confirmed by the Department of Pathology, Medical College, Agra. Of these 37 were diagnosed by positive agglutination test alone and 19 by positive blood cultures.

Table I—Showing Distribution of Enteric Cases According to Sex and Age

Total No. of Cases	Males	Females	Age in Years 1-10	11-20	21-30	31-40	41-50	51-60 & over
56	37	19	12	12	25	6	nil	1

Table II—Showing Results from Different Types of Media for Enteric Infections

	Glucose Broth	Gentian Violet Taurocholate	Bile Alone
Positive	.. 3 (9·4%)	19(58·4%)	5(50%)
Sterile	.. 24	17	3
Contaminated	.. 5	nil	2
Total	.. 32	36	10

Blood culture as a routine in taurocholate gentian violet media was used for six months only and during this period 36 cases of enteric fever were examined. Previous to this glucose broth in 100 c.c. amount was used and to this 10 c.c. blood was added. Positive blood cultures were only found in 3 out of 32 cases examined, giving the figure of positive 9·4 per cent. Pure bile alone was also used in blood cultures and its results are as encouraging as those of taurocholate gentian violet (Table II). In clot culture it does not prove to be as efficacious as the combined bile salt gentian violet media.

Out of the 120 total blood cultures done for all types of cases during the one year only 24 have proved positive (20 per cent), and even these were seen when the incubation was long. The earliest that a correct diagnosis was made in enteric fever cases was after 36 hours of the inoculation of the media and latest after 6 days had passed (Table III).

Table III—Showing Frequency of Positive Cultures after Primary Inoculation of the Media

Time after inoculation in hours	Number of positive cultures
24	nil
36	3
48	6
72	4
96	3
120	2
144	1

The distribution of the various members of the salmonella at Agra as found by the author is given in Table IV (55 cases).

Table IV—Showing Distribution of Various Members of Salmonella

Organisms	Whole blood & clot culture	Agglutination test	Total
Bact. typhosum	.. 17	22	39(69·7%)
Bact. paratyphosum A	.. 1	12	13(23·2%)
Bact. paratyphosum B	.. 1	2	3(7·1%)
Bact. paratyphosum C	.. nil	nil	nil
Bact. enteritidis & others	.. nil	nil	nil

Mostly the causative organism is *Bact. typhosum*, and *Bact. paratyphosum* A and B come second and third in importance. So far no case of *paratyphosum* C or any other type of salmonella has been isolated, but one has to be on the lookout for these as infections by them have been reported in this country.

Table V—Showing Duration of Illness When Blood Cultures were Done and Found Positive

Duration of Illness in days	Whole blood culture	Clot culture	Total	Sterile	Grand Total	% Positive
1-7	8	0	8	0	8	100
8-14	3	5	8	2	10	80
15-21	1	0	1	6	7	14·3
22-28 & after	0	0	0	9	9	0
Relapsing cases	1	1	2	0	2	100
Total	13	6		17		

Conclusions

1. The freshly isolated strains of *Bact. typhosum* are often inagglutinable or slightly agglutinable. These on repeated subcultures become agglutinable.

2. With the media used the appearance of typhoid or one of the paratyphoid bacilli is seldom delayed for more than 48 hours, growth in the majority of cases can be detected in 24 hours, so that the identification is complete within 48 hours of taking the blood.

3. Blood culture from cases of suspected enteric fever should be kept for 7 days and examined daily before discarding. If at the end of one week incubation growth is not obtained, discard the bile or

taurocholate cultures but keep the remaining ones for full one month to see if other organisms will develop.

4. The occurrence of mild and transitory infections due to typhoid organisms which may produce chronic carriers make it essential that blood culture be made immediately there is the least suspicion that a patient is suffering from enteric fever.

5. Enteric fever is to be considered a possibility in cases of abortion or premature labour associated with fever, as pregnant women who contract this disease are specially liable to these accidents. Enteric fever may also appear shortly after delivery. Blood culture often elucidates the cause of such fever and this happened in one case who was investigated for puerperal sepsis but actually proved to be suffering from an attack of enteric fever.

6. Provided suitable methods of cultivation are used a negative blood culture at the beginning of a febrile illness makes it unlikely that the disease is enteric fever.

7. Blood culture though negative in latter period of the illness again becomes positive in relapses.

8. Bacteraemia in paratyphoid infections is of short duration and cultures done even on the third day of the illness have proved negative.

9. No true relationship of positive cultures in unvaccinated persons to those in vaccinated ones could be obtained.

10. Most of the patients are taken to the hospital in the second week and few only in the first week. Thus it is for the practitioners who are looking after the treatment to resort to blood culture at the early stage of the disease. So far the aid of the pathologist was not possible but now with the establishment of laboratories at the district head quarters it should not be the case. Most of the cases in towns and cities, not to speak of rural areas, remain undiagnosed and become sources of the spread of the disease in isolated localities. Thus from the preventive aspect, blood culture, if properly done, forms a very essential step.

Acknowledgment

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MACROCYTIC AND DIMORPHIC ANAEMIA IN INDIAN TROOPS

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Forty cases of advanced anæmia admitted in 48 I. G. H. in Ceylon, were observed during the period from September 1942 to March 1943.

The study of the morphology of the cells was based upon the latest descriptions of haemopoiesis by Turnbull (1936) and Israel (1939) further elucidated by Trewell (1943) who worked on a large number of cases of dimorphic anæmia in Uganda in 1941.

The dimorphism resulting from dual-deficiency in the present series of cases was detected by (1) cytological study, (2) double reticulocyte response, and (3) response in general blood picture and clinical condition.

Excepting the few advanced and serious cases of liver-principle deficiency the rest were put first on iron and ascorbic acid. As a rule there was a reticulocyte response and rise in the blood counts. But after a few days the former subsided and the latter had a halt when the substitution of liver principle in the treatment was accompanied by a second reticulocyte response and brought the blood to the normal level. A few cases resistant to iron responded to liver-principle first and then to iron. The few advanced cases responding well to initial treatment with liver-principle had also secondary response with iron in the later stages.

Out of 40 cases, 21 had their colour-index above 1·3. Dimorphism was detected also in the group below this colour-index, but the present observations were mostly limited to the group with colour-index above 1·3.

The diet among the troops was satisfactory and could not account for any deficiency in hæmatinic principles.

A thin blood film was found satisfactory to study the type or types of erythropoiesis and the percentages of macro and microcytosis.

Notable Features and Discussion of the Cases

The first group (with high colour-index and predominant macrocytosis in the films)—There were two cases of chronic malaria with marked enlargement of the spleen extending downwards below the umbilicus. They resembled cases of Bengal splenomegaly. They responded well to liver. Their blood remained at the normal level for two months during their stay in the hospital after recovery. In one case the spleen contracted very well and came up to 3-fingers below the costal margin.

Two other cases were gravely ill and highly anæmic at the time of admission. One of the cases was running a high remittant temperature. He had no history of malaria, the colour-index was 1·6, red blood cell count 0·9 million, and the film showed predominantly large number of megalocytes and macrocytes with fair eosinophilia (haemoglobinisation) of cells. The nucleated red cells in the film were few (only 2 per cent of the leucocytes which were 2600 per c. mm.) and they corresponded to late megalo-

blasts of Israel. The gastric juice showed low acidity. No parasites were detected in the blood film. There was no enlargement of the spleen. The patient had hookworm infection. The eosinophilic polymorphs were 17 per cent or 442 per c. mm. The neutrophil count was 1170 per c. mm. Before admission in the writer's ward, the patient was given 2 courses of hookworm treatment followed by a course of iron for 43 days. Obviously there was no response.

This case responded to intensive dosage of liver given intramuscularly within a week, and after that the improvement was fairly rapid with further treatment with liver. His final blood count on the sixty-second day was 4·5 million and Hb 75 per cent. He was also given blood-transfusion after his response to liver.

The rest of the cases of orthochromic anæmia with high colour-index numbering 17 showed predominant macrocytosis of the red cells and 11 amongst them showed definite dimorphism and dual-deficiency. The deficiency of the liver-principle was more marked. In most of them a few nucleated cells were found in the film. The cells corresponded largely to normoblastic erythropoiesis.

Two of the fairly advanced cases of anæmia had no response to iron treatment in the initial stages. They had malaria infection but no hookworm infection. They commenced to recover when the liver treatment was started, and later on it was supplemented by iron with advantage to make up the deficiency.

Six of the other cases had only slight response to iron used for a long time at the first hand, but later on their response to liver was satisfactory. Weak primary response with iron and a marked secondary response with the liver given later on was a common feature.

Most of them had history of malaria, but only in three cases parasites with corresponding changes in the blood were found.

Six of these cases, particularly the bad ones had no enlargement of the spleen, and the rest excepting two had very little enlargement The enlargement of the spleen in requisite proportion to the disease is only a happy augury as sign of immunological proliferation of the reticuloendothelial system. In some cases, the disease seemed to have advanced rapidly and turned serious without proper treatment and rest while the spleen could not keep pace with the disease. The experience in these cases was that the cases with proportionate enlargement of the spleen responded better to treatment than the cases without enlargement.

Summary

(1) One case of macrocytic orthochromic anæmia with high colour-index of *hookworm infection* was observed. This case did not respond to iron and the unloading of worms. There was a fair response to liver treatment. Partial aplasia of the bone marrow with slight megaloblastic erythropoiesis was evident.

(2) One case of macrocytic orthochromic anæmia with high colour-index of chronic and latent *malarial infection* was observed. The iron deficiency was very little and masked. Predominant megaloblastic with some normoblastic erythropoiesis was the marked feature in the thin films. The case resembled the achrestic anæmia of the macrocytic type described by Israel.

(3) Some cases of chronic or subacute malaria with or without hookworm infection showed dimorphism with more marked deficiency of the liver-principle. They were cases of macrocytic orthochromic anæmia with high colour-index. A suggestion has been made that dual deficiency may supervene whenever any anæmia due to a single etiological factor advances. Usually they had weak and unsatisfactory response to iron given at the first hand. All of them had good response to liver, and in a few cases slight response to iron was observed only after the major deficiency *i.e.*, liver-principle was supplemented. Double reticulocyte response was elicited in most of the cases. The *erythropoiesis in general in thin films of blood was normoblastic.*

(4) The absence of enlarged spleen in advanced anæmia of malarial origin did not present a happy spectacle. Such cases had darker outlook and poorer response to treatment than the cases with proportionate enlargement of spleen.

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(*Concluded from page 121*)

(3) Sulpha drugs are usually not effective in staphylococcal septicæmia and penicillin should be started early. The interval between penicillin injections should not be more than 3 hours.

(4) In the latter part of the disease the persistence of temperature is due to suppuration in some of the muscular swellings. Parenteral penicillin has no effect on localised pus and the abscesses must be opened up and properly drained.

(5) Penicillin has only a very temporary bacteriostatic effect and allows the body defences to increase and act on the static organisms. The immune bodies can be increased in the later part of the disease by the staphylococcal vaccines.

(6) The patient should be carefully watched after the penicillin therapy is stopped for there is always a danger of relapse.

My thanks are due to Dr. N. Subhie, Dr. Man Singh, Dr. B. N. Kapoor and Dr. S. N. Mathur for taking notes from their cases.*

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CAVITIES IN PULMONARY TUBERCULOSIS

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The most important role in the drama of pulmonary tuberculosis is enacted by cavities. Had it not been for their presence, half the fear and dread of this terrible scourge would vanish, and through the darkness of despair would creep in a ray of hope. It is a subject which confronts the pthysiologist in his every day life, giving him food for further thought of conquest of this intractable disease. How often have we found the best of our treatment fail to close the cavity with its disastrous consequences and results. Hence the importance of collapse therapy cannot be too highly emphasised. In our land where this terrible disease is rife, poor and ignorant patients knock at our doors for treatment when far advanced with multiple thick walled cavities. Every day in the life of a cavity counts, and with its horrible consequences, it should be our bounden duty to tackle it as soon as discovered, and to use the best remedies to bring about its closure. Every cavity is born, grows from stage to stage, and ends up as a thick walled fibrotic space. This is the rock on which the ships of collapse therapy founder and are destroyed. If only we could get these cases early!

I shall deal with this subject under various headings, thus facilitating division of labour, and at the same time simplifying its digestion.

Pathology

It was known to the Hippocratic school. Cavities are the end results of caseation and proliferation. To start with tubercle bacilli are brought to the lung, and depending on the number, virulence, and tissue sensitivity, cause either exudative or proliferative changes. Cell necrosis with polymorph infiltration is the first step. It gives way to serious exudation, *viz.*, W.B.C., R.B.C., plasma, fibrin, and later on mononuclear and mesenchymal cell infiltration with formation of epithelioid cells, giant cells, etc. This mass gets surrounded by a layer of lymphocytes and fibroblasts. Exudation undergoes fatty degeneration, with conglomeration into a homogeneous mass, namely, caseation. This material imbibes fluid and acted on by polymorphs undergoes liquefaction, which when rupturing through a bronchus gives the birth of a cavity. This cavity at first has no wall of its own, being an emptied space from extrusion of the recently contained necrotic mass with ragged walls. With time it gets surrounded by a second layer of tubercular granulation tissue and finally completes maturity being three layered. The inner or pyogenic membrane, composed of necrotic tissue, pus, blood, fibrin, tubercle bacilli and obliterated and thrombosed vessels; the middle coat of tuberculous granulation tissue; surrounded by an armour of young fibrous tissue.

Growth of a cavity takes place by (*a*) destruction of the surrounding tissue, (*b*) fusion of adjacent foci of suppurative liquefaction, and (*c*) by ballooning due to a check valve bronchus without tissue destruction. It has a tendency to spread in the caudal direction, due to the macerating action of the pus at the bottom.

The bronchus plays an important part in the growth of a cavity. It undergoes inflammatory changes, congestion, ulceration, caseation, tuberculous granulation and fibrous stenosis, thus giving rise to the formation of the three different types of valves, *e.g.*, by-pass, check-valve, stop-valve. The bronchus opening into a cavity may do so either at the bottom, when usually the cavity is dry, or may open on the lateral wall as visualized on the x-ray with a small puddle of fluid at the bottom having a horizontal upper level, or lastly may open on the top when the cavity is generally full. Most tubercular cavities being situated over the upper half of the lung with bronchi opening at the bottom are dry ring shadows unless there is a check valve mechanism. Compared to the above pneumonic and secondary abscesses which develop over the lower half of the lung field, are generally full.

Pathological Physiology—For this we are greatly indebted to the classical work of Coryllos. His enlightening analysis of these problems brings us closer to the understanding of various phenomena we come across, when studying the behaviour of cavities. Most cavities after their emptying out get walled off, and form an irregular jagged appearance and take on a more circular shape. Coryllos explains this by the difference of gas pressure within the cavity and the surrounding lung parenchyma. The air entering through the patent bronchus is at atmospheric pressure, whilst that of the surrounding lung parenchyma, permeated with tuberculous infiltration is at negative pressure. The difference accounts for the cavity assuming its circular shape. The draining bronchus plays an important part in the growth and obliteration of cavities, which I shall deal with later on under the heading of healing of cavities. As soon as the above by-pass valve becomes a check-valve, due either to mucus, œdema of the mucous membrane, tuberculous granulation tissue, the cavity now enters the stage three. As the air entering cannot easily leave, the cavity grows by ballooning out without parenchymal destruction. Such check-valve cavities grow to huge dimensions, and at the same time have a tendency to vanish as soon as the check-valve becomes a stop-valve. We have seen fair-sized cavities vanish with mere bed rest, and which can thus be easily explained, although this is a rare phenomenon.

The disappearance of the cavities depends on (*a*) exchange of gases between the air within and the vascular wall of the cavity with the surrounding lung parenchyma, (*b*) thinner walls made of granulation tissue and the better the circulation, the more rapid the collapse, (*c*) the surrounding parenchyma being less infiltrated offers less mechanical resistance to collapse, and hence the more normal the parenchyma the quicker will be the collapse.

The part played by the bronchus:

(1) Bronchi expand and contract with respiration.

(2) Collapse therapy by distorting its lumen and obliterating it, brings about a stop valve, with absorption and disappearance of cavities. This phenomenon is akin to atelactasis following bronchial obstruction.

(3) By pass valve with circular cavities. Check-valve with balloon cavities. Stop-valve with disappearance of cavities, or patent germ free cavities.

(4) Tension cavities resulting in failure of treatment to artificial pneumothorax (they may even continue to grow), and even to thoracoplasty. Herein the bronchus is partly occluded and stenosed.

(5) Infiltration of the bronchus with tubercular involvement produces check-valve cavities, atelectasis, tension cavities and secondary infection with grave prognosis.

(6) Easier collapse of cavities situated away from the hilum, as their thin elongated bronchi can be easily collapsed and kinked.

(7) When an adhesion is held taut in the line of the draining bronchus thus keeping it open, no amount of A.P. pressure will collapse the cavity.

(8) When the adhesion is vertical suspending the cavity, phrenemphraxis aided with pneumoperitoneum is able to bring about a kinking of the bronchus and obliteration of the cavity. When the adhesions are horizontal, no amount of elevation can bring about a kinking of the bronchus.

Healing of Tubercular Cavities

There is always a controversy between the clinical and pathological healing of cavities. To the former the x-ray picture shows the disappearance of the ring shadow, replaced by an opacity with negative sputum; whilst to the latter such is not the case in the true pathological sense; as T.B. are still present in the collapsed walls which are composed of tubercular granulation tissue. Two methods of healing claim our recognition. The closed which is the commoner and the open.

The first or the closed method.

(1) When the check-valve becomes a stop-valve (plug of mucus, œdema, granulation tissue, fibrosis, and contracture of scar tissue at the cavity-bronchus junction), the air inside the cavity gets cut off from the exterior. Gaseous interchanges take place between the air inside and the vascular atelectatic tissue in the wall and surroundings, and cause the air within to be absorbed. The cavity begins to shrink in size till it completely disappears. The granulation tissue comes into apposition, fuses, and being invaded by fibroblasts and connective tissue gets organized into a firm scar. Such is the healing seen in the by-pass and check-valve type of cavities when they get converted into the stop-valve variety. At times rest alone causes such cavities to vanish, the credit of success going to gold or tuberculin or any other medicinal line of treatment adopted. These are the type of cavities that disappear to reappear within a short time, the valve opening out, once more becoming by-pass or the check variety. Where collapse therapy has been tried, the stop-valve has been brought about either by distortion of the bronchus, or by external pressure obliterating it.

(2) In certain cases the walls although they fall in fail to appose, leaving a potential space between. Roentgenologically we see an opaque scar. These same scars open up when the compression is discontinued, and once more shape themselves into a cavity.

(3) In the types of cavities classified as (1) and (2) the walls appose by different collapse treatment, granulation tissue come into contact, infiltrate each other and fibrose later to form a scar.

(4) In this variety the pyogenic membrane gets shed off. Granulation tissue gets invaded by fibroblasts and becoming converted into fibrous tissue starts contracting. As it contracts the lumen gets smaller and smaller, until firm scar tissue has obliterated the space. Herein the bronchus plays a minor part. Such is the type of healing seen in cavities type 4 & 5.

(5) In this variety the bronchus gets shut off. The lumen of the cavity space gets filed in with caseous material from the pyogenic membrane, thus being completely obliterated. This mass gets inspissated and later on calcified. The walls become transformed into fibrous tissue. Such are the clinically healed yet pathologically active cavities, since the caseous and calcified material still contain and harbour viable tubercle bacilli for years. They are very often hidden volcanoes which burst out later with disastrous results.

(6) Emphysema of the surrounding lung tissue has been attributed as one of the causes which compress the walls of the cavity and bring about their healing.

(7) The elastic recoil of the lung has been attributed to keeping the walls apart, and on its relaxation by A.P. or phrenemphraxis brings about the collapse of its walls.

The second or the open method of healing.

It is generally found in types 4 & 5. As a result of the thick fibrotic incollapsible wall, apposition is not possible. The inner lining of the cavity, the pyogenic membrane sheds itself throughout the patent bronchus. The wall now comes to be composed of tubercular granulation inside and a thick fibrotic wall outside. The granulation tissue comes in time to fibrose and thus heals itself. The mucous membrane creeps in through the bronchial opening, and spreads over the wall giving it a clean new lining. The mucous membrane is composed of squamous or columnar epithelial cells. The above is the explanation for thick-walled cavities with patent bronchus which show a persistent negative sputum. As Sorgo states, this type of cavity is more a defect than a disease. In the Monaldi's treatment of the drainage of the cavities the aim is to bring about a similar type of healing of the cavity.

Roentgenological Classfication of Cavities

Type 1.—Early acute cavity formed as a result of softening and sloughing out of caseous material. Walls are ragged and composed of destroyed pulmonary

tissue. May be single or under hyperallergic conditions multiple. Radiologically they have no walls being seen as an irregular patch of rarefaction within a dense opaque area.

Type 2.—Early progressive cavity. A definite wall of granulation tissue has been formed. By progressive ulceration outwards it enlarges growing bigger and bigger. A definite circular shadow is seen with granulation tissue in its walls. The inner lining is irregular depending on the excavating process. The bronchus is of the by-pass type, and hence these cavities appear round. On inspiration and expiration they enlarge and contract as air passes freely in and out.

Type 3.—Check-valve variety. Here again the wall is formed by granulation tissue. The bronchial opening plays the part of check-valve caused either by mucus, congestion, or granulation tissue. Regular increase in size without destruction of the surrounding lung parenchyma helps to differentiate it from the above. It gradually balloons out. The check causes fluid to accumulate at the bottom forming a puddle. Clinically it is of the silent variety. When as is not infrequent the draining bronchus is visualised due to tuberculous bronchitis, the cavity is designated as the stem cavity or the tennis racket cavity.

Type 4.—Non-progressive smooth-walled fibrotic cavity. The wall is formed of granulation tissue and surrounded by fibrous tissue. The bronchus is patent.

Type 5.—Thick-walled fibrotic cavity. Very resistant to collapse therapy if the draining bronchus is occluded. This is the worst type of cavities for closure by any method.

Type 6.—The last but not the least important is the bronchiectatic variety seen mostly in fibrotic retracted tuberculous lungs. These are best visualised by lipiodol, the ordinary plate showing little, the lung being fibrosed and thickened, and so the pleura.

The first three varieties with granulation tissue as their walls are very amenable to treatment. The latter due to their thick fibrotic walls are resistant. These are the types of cavities that we as medical practitioners should try not to see, and hence the value of treatment in early tuberculosis.

Situation of Cavities and their Importance

Most of the cavities are seen in the infraclavicular zone. In type 1, 2 and 3 in the middle of the lung, away from the pleura, collapse therapy gives the best results as there are few adhesions. The nearer the cavity is to the pleura the more chances of the adhesions, and the permutation and combination of various collapse therapy to bring about its collapse. Where single adhesion is perpendicularly holding up the cavity, phrenic relaxation will bring about its close. When at right angles, internal pneumolysis will have to be done. Where part of the cavity wall is adherent to the pleura both visceral and parietal, and where A.P. has failed, phrenic relaxation will do no good. This requires a thoracoplasty.

In cavity type 4 artificial pneumothorax gives the best results. Where the cavity is suspended by adhesions phrenemphraxis fails to obliterate the cavity its walls being fibrotic.

In cavities type 5 and 6 thoracoplasty is the only main stay and at times it may even fail.

Where cavities are situated in the mid zone collapse therapy gives the best results in type 1, 2 & 3, and the worst type of results in 5 & 6 and also in tension cavities with stenosis of the bronchus. Parahilar cavities at times fail to respond to A.P. and phrenic, and have to be collapsed by thoracoplasty.

Basal cavities type 1, 2 & 3 respond well to phrenic and artificial pneumothorax.

Complications

I shall just state them in brief in order to show their importance in the life chapter of a tuberculous individual, and stress the seriousness of their nature. Every day in the life of a cavity counts. It is the sword of Democles which hangs by a thread, and that too a worn out one. One hæmoptysis, contralateral spread, or spread to other parts of the body, *e.g.* the meninges, and the case from the crest of hope passes down into the vale of disaster. The complications we come across are the following:

(*a*) Local spread in the same lobe, and from lobe to lobe, and from lung to pleura with subsequent pleural developments. (*b*) bronchial spread with seeding implantation in the same or contralateral lung, (*c*) lymphatic spread in the same lung or to the glandular components or to the general grandular tree, (*d*) vascular spread with involvement of other organs and miliary, (*e*) hæmoptysis being fatal, or leading to spread in the same lung or contralateral spread, (*f*) involvement of the bronchi, trachea, and larynx with resultant failure of treatment, (*g*) secondary infection to the intestines.

Site of Aspiration Foci.—(*a*) Lateral aspect of base of upper lobe, (*b*) apex of opposite lung, (*c*) middle and lower lobes, (*d*) apex of the lower lobe, (*e*) base of the lower lobe.

Clinical Findings

The physical findings of cavities given in the different text books are not in themselves infallible criteria for diagnosis. How often to our horror and shame we find the x-ray speak volumes when the stethoscope is dumb. In short may we say that there are silent cavities and those that speak. The cause of silence may be attributed to two important factors, the thoracic cage and the lung proper. In the aged the rigid chest wall with emphysema, or even in the young with barrel-shape chest it is at times very difficult to locate anything. Inspection and palpation help very little and even percussion in such cases fail. Auscultation only helps when the intervening lung tissue is pathological, conducting sounds or when the cavity is very superficial. The cavity itself may not speak when it is of the check or stop valve variety.

Even in those that speak, inspection and palpation teach very little. In percussion we have localised dullness or very rarely tympany if the cavity is very large and superficial. In auscultation we have bronchial, tubular, cavernous and amphoric breath sounds with bronchophony and whispering petrology, in cavities type 4, 5 and 6. Cavities type 1, 2 and 3 are mostly silent. Foreign sounds with bubbling and

metallic rales are also found. In spite of all these clinical findings the x-ray still holds the centre of the stage.

Treatment of Cavities

I shall not go into the treatment in detail as it would itself take up a full chapter. I must say that today surgery stands aloof from medicine because of the fact that we allow the disease to progress from its early treatable stage to the stage in which it has to be handed over to the surgeon or to his creator.

So long as we continue to allow cavities to progress from stage to stage and leave stage 3 behind, so long will we have to pass our cases on to the surgeon. The medical practitioner in India today forms the soldier in the firing lines. It is on him that we have to rely for an early diagnosis. If he can tackle the cases from the very start, or send them on to the institutions better equipped, I am sure that cavities can be halted at this stage 3. We may do whatever we like in the programme of tuberculosis advancement, but I must say that we still have to rely to the utmost on the medical practitioner.

Cavities 1, 2 & 3.—In the treatment of these cavities *gold* plays no part. Gold seems to be the mainstay of the private practitioner, and to him I will boldly say that to treat a case of tuberculosis with cavitation on gold is to condemn the patient to certain death. Gold never healed a cavity and never will. *Rest* alone will heal cavities belonging to the check-valve variety. I have seen this in a very few cases, but I must say that to rely on rest alone is to play with fire. *Artificial pneumothorax* has been our faithful friend since the beginning, and we can rely on it with full confidence. I am all for artificial pneumothorax being started at the earliest for then we get the best results. *Phrenemphraxis* when done for the above has given us very good results. We have used it alone, or combined with A.P. or with pneumoperitoneum. The earlier the stage in development the surer will the diaphragmatic relaxation give beautiful results, and for check-valve cavities it has given remarkable results. We have had a lot of experience in *pneumoperitoneum* and where A.P. has failed phrenic with pneumoperitoneum has given fair results. *Internal pneumolysis* is of value as aid to artificial pneumothorax to bring about better collapse and obliteration of cavities.

Let this stage pass and we drift into the realms of major surgery. I shall not go into details of various operative procedures but suffice it to say that modern *thoracoplasty* today is our main stay for types 5 & 6. The cavity type 4 is the one that frequently behaves like 1, 2 and 3 and quite often like its opposite component 5 & 6. *Monaldi's treatment* is meant for thick-walled cavities type 5 where thoracoplasty cannot be performed.

May I conclude with the prayer that in the near future a drug may be born that will be specific for this wretched disease, and then goodbye to cavities, the dread of pulmonary tuberculosis.

Acknowledgment

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STAPHYLOCOCCAL SEPTICAEMIA

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Staphylococcal septicæmia usually occurs secondary to some recognisable focus, but in these four cases which I recently observed no primary focus could be discovered. All these cases started with fever and painful swellings in the muscles and bones. Except in one case they did not show any marked toxæmia. Total white blood cell count ranged between 15,000 and 20,000 per c.mm. with 80 to 90 per cent neutrophil polymorphs. Curiously two of these cases showed an eosinophil count of about 10 per cent. Blood culture showed staphylococcus aureus in two cases. In one case it was sterile but a high white cell count, persistent swellings and marked relief under penicillin treatment was sufficient for diagnosing a septicæmic condition. Moreover, the blood culture was taken when the patient was receiving sulphathiazole which exerts a bacteriostatic action in blood culture and results in sterile reports. In the fourth case no blood culture was done, but the typical history and the presence of staphylococcus aureus in pus from abscess strongly confirmed the diagnosis of septicæmia.

Treatment in all these cases was first by cibazol and later by penicillin. Dosage of penicillin varied with severity. In the severe case the three-hourly injections were continued for 10 days. In this case the early cibazol therapy depressed the total white cell count to 10,000 per c.mm. with 84 per cent polymorphs and one started doubting the diagnosis of septicæmia and suspected enteric. Penicillin was started to avoid any risk, but the diagnosis was confirmed the next day by blood culture which showed pure growth of staphylococcus aureus and the presence of a coagulum in the glucose citrate broth medium indicated that the organism was of pathogenic variety. Under penicillin treatment all these four cases were cured. Most of the muscular swellings subsided, but some developed into abscesses and had to be opened. The temperature persisted till all pus pockets were drained. It was no use continuing penicillin with unopened abscesses. In one case low temperature persisted after all the abscesses had healed up. The patient was given as many as 80 tablets of cibazol without any effect on the temperature. One injection of 100 million organisms of stock staphylococcal vaccine was given. The temperature rose upto 102°F. but fell down to normal the next day and did not rise again. Gradual doses were given to build up further immunity. In these observations the following points are worth noting:

(1) Septicaemia should be suspected in cases showing fever and swelling in the muscles. Some of these cases are likely to be treated as enteric.

(2) Blood culture should be done early in not less than 100 c.c. of glucose citrate broth. If the patient is receiving sulpha drugs the broth should contain 5 mgm. per cent of para-amino-benzoic acid in order to counteract their bacteriostatic action.

(Concluded on page 117 at foot of col. 2)

BONE GRAFTING

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Introduction

Of all the types of tissues in which graft is attempted bone grafting has the largest field of usefulness. In economically poor countries more attention should be paid to bone grafting to enable the unfortunate patients to return to their wage-earning capacity. The idea of the present paper is to interest a larger section of our surgeons in bone grafting by showing how easily satisfactory result can be obtained.

Reference to the practice of grafting tissues in this country is very old. It dates back to the Hindu mythological era where mention has been made in the Ramayana, of testicular grafting and of transplantation of human rib as well. In the latter part of the 19th century, bone grafting came into much prominence. The practice of bone grafting has gained considerable impetus with the pioneer work of Albee since 1904.

I will confine myself to massive cortical bone grafting. Majority of surgeons prefer tibia as the donor. I have exclusively used fibula. Fibular grafts present certain interesting features of their own. Fibular graft is a very handy material. You can shape it diversely. One can make screws, nuts or nails out of it. One can chop it up or use it whole depending on the varying needs.

A great deal of controversy has arisen with respect to the part the graft plays. The arguments put forward are whether the graft affords but a scaffolding material or does it become the integral part of the recipient. Opinions are widely divergent on this point. Unfortunately the evidence gained from results of operation does not enable us to come to a definite conclusion.

As for me, I believe that the fibular graft does not die. This I assert on the basis of my own experience of my follow-up cases. The graft is taken up. Nature makes a real attempt to make up the superstructure on the skeletal foundation frame work afforded by the graft. The mechanical conditions being favourable the graft becomes an integral part of the recipient. The amount of mechanical stress and strain, the direction of the component forces and the mechanico-physiological requirements of the local area concerned together with the availability of favourable environment, I think, determine the future fate of the graft. The real answer to the fate of bone graft depends on what use you want to make of it and what is nature's need at the given site. If the graft is put across a mobile joint, where nature wants movement, you will find that there will be effort at absorption of the graft. On the other hand where transmission of weight is called for, nature strengthens your effort and the graft grows. Strange as it may seem, it often tends to acquire the original shape of the part where it is grafted (well brought out in my case of grafting for the lower end of the radius).

Technique

In the actual technique three points are to be stressed, *viz.*, asepsis, apposition and immobilization. For control of sepsis in cases of doubt (?) I used penicillin and sulphathiazole, locally as well as parenterally. I have made no great attempts to secure intimate accurate apposition, on which point others have laid great stress. To obtain absolute fixity and immobilization most workers use autogenous bone pegs or heterogenous bone pegs or vitellium screws and wires. I have used catgut alone to ensure fixity. Yet all grafts have taken and have been serving well. I have depended upon external fixation by plaster of Paris and paid meticulous attention to change it before it was too loose to serve the desired purpose. Plaster fixation was maintained from three to four months in most cases, while internally the graft was kept in position by catgut. The satisfactory successful results obtained speak for themselves. The only type in which I have failed were cases of septic tubercular arthritis with sinuses discharging pus. Attempts to fix these joints by criss-cross fixation of bone to limit the course of the disease, unfortunately failed, though Albee has been successful with several such cases. I found bone grafting very useful in cases of benign tumors. In one case of myeloma of the lower end of radius, the lower radial end was excised. A fibular graft was put in to make up the deficiency. The graft was split to resemble the lower end of radius. The graft has taken and grown. Nature hae attempted to reshapen the graft and to mould it into its usual normal form.

Another case was of fibrocystic condition of tibia in which the whole shaft was involved. It was excised. The shaft was reconstructed with fibular graft. The graft got consolidated and became fused with the original tibial ends. Subsequent radiography proved that the graft lived.

The next case was that of a myeloma affecting the neck of the femur. The problem before me was whether to amputate or to reconstruct. I decided in favour of the latter course. A lateral approach was made. The neck was excised and carbolised. Two massive grafts were put in which grew and remodelled themselves into the shape of the original neck and became strong.

My further efforts were directed to immobilisation by internal osseous splintage in some cases of spinal tuberculosis, where secondary infection had not supervened.

In cases of non-union, of course, bone grafting alone offers the hope to enable such incapacitated persons to return to normal life. A lady who had the misfortune of fracturing her forearm four times during the course of 20 years till the limb became flail consulted me. It was difficult to persuade her to undergo operation especially when she was told that a fibular graft would have to be made use of from her own leg. An inlay bone graft was put in with perfect result.

Next was a case of non-union of fracture of the neck of femur in an 11-year old individual. A bone graft was put in with successful result.

Lastly, recently, I have tackled a case of non-union of fracture of tibia. I used the companion fibula as an only graft to bridge the gulf of its fractured partner following Bohler's aphorism that "when tibia goes ununited excise the fibula."

(Concluded on page 131, col. 1)

SPECIAL ARTICLES

I

INDUSTRIAL HEALTH—A PROBLEM FACING INDIA

Some Needs and Some Suggestions

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India stands today on the threshold of extensive industrialization and her people rightly look forward to greater industrial employment and increased productivity. Out of a total population of nearly 400 million less than 20 million are dependent on industry for their living and only some 2½ million people (*i.e.* 0·6 per cent of the population) are actively engaged in large scale and regular industrial establishments. (Chakravorti, 1946).

The potential resources for industrial development of the country are great but with the present underpaid, underfed, badly housed and unhealthy population the outlook is disconcerting. While it is high time that more and more people were brought in to play their parts in the industrial life of the country, so far there has been little evidence to show that Indian industrial organizations as a whole are applying, on any demonstrable scale, the results of research into the "human problems of work". The industrial worker is as important as the machine, and the "health protection of the man who works" is of paramount importance (Wampler, 1943). If India must avoid those heavy losses in men and material which accompanied the industrial revolution of the West, the management of Indian industries must harness the knowledge and experience of their counterpart in the United Kingdom, the United States of America and the Soviet Union. There was a time when nobody seemed to feel any responsibility towards the health and welfare of the workers; neither the employer nor the State took any serious trouble to inquire if the workers were physically fit for their jobs or whether harmful conditions of work were adversely affecting their health. The result was that many people of "occupational age-group" broke down in health and if they did not die prematurely, certainly, they eventually became a burden on the nation. No progressive country to-day can afford this preposterous loss in "man-power-hours".

The fact that ill-health of the worker causes not only a loss to industries but is an ultimate drag on the nation has come to be appreciated by comparatively few organizations. A tremendous amount of research work has been done both in England and in America into the effects of hours of work, of conditions and environment, the cause and control of accidents, and a host of other problems relating to industrial health. Above all, the important task of personnel selection, "finding the job for the man and the man for the job" has received a great deal of public attention in the more advanced industrial countries. To wit: some people are physically and mentally unsuited for certain types of work, especially in the chemical and mining industries (*i.e.* people who are prone to industrial dermatitis and respiratory diseases etc.); on the other hand, certain categories of visual acuity and "visual grades" are especially suited for fine work in precision-instrument making. Such examples could be multiplied manifold. A misplaced worker, "a square peg in a round hole", is not only a loss to the firm; he is unaccommodative to the environment of his work and to his colleagues, and is, in fact, just the subject to become "accident-prone".

This factor of "human element in industry" appears to have received very little attention in India. Pre-employment medical examination of prospective employees and a carefully planned system of allocation into suitable types of work are necessary if man-power is not to be wasted (Smith, 1943). The responsibility of the employer does not end with "owning a factory", nor can the labour manager sit back when he has supplied his foremen with so many hands to tend the machines. The worker must of necessity spend a third or more of his daily-life in a factory or workshop; it is not too much then to expect that his welfare and health problems should become a major concern of the management and the State.

The still too prevalent practice on the part of the employer to dismiss a worker the moment he falls ill or is unable to carry on the job he is set to do is to be deplored, especially when his illness may probably have been due not so much to his own constitutional health but in part due to a fault in the process or the machine. The Statistics of Factories, published annually, give figures for "accident in factories" but no reference is made as to the cases of sickness from toxic hazards or other occupational risks. The reports of the mines do not speak of any incidence of pneumoconiosis or the cotton mills of byssinosis though it is probably fair to assume that cases do occur. It is both more profitable and productive to train a person for a particular process and aim at getting years of useful service from him rather than sack him after the first few months because he is suddenly taken ill. Very often a transient illness is the cause of permanent loss or diminution in the earning capacity on the part of the worker, for, unfortunately, so long as labour is so cheaply obtained in India, the employer finds it an easier task to replace the worker who has taken ill rather than investigate and remove the cause.

Apart from the few pioneers of large-scale private industrial enterprise, the Government of India is to-day the biggest single employer. And it is gratifying to note the efforts of the centre to pursue a progressive and enlightened policy with regard to the health problems in industry. This is manifested by the recent intensification of the Factory Inspectorate System and also by the visit of some of its personnel to the United Kingdom to study labour problems and the methods in the application of the Factories Acts. But here too a large gap wants yet to be bridged. So far very little attempt has been made to incorporate sufficient medical personnel in the Factory Inspectorate on similar lines as the Medical Inspectors in the H.M. Chief Inspectorate of Factories in Great Britain, and it is hard to believe how "safety and health measures" could be effectively enforced and adequately supervised in India without the assistance of medical guidance.

There can be no doubt that a medical man is best fitted, under the light of modern conditions, to assess the clinical aspects of industrial hazards and toxic processes, and that his knowledge and sympathetic co-operation is of vital importance to any industrial health project.

Admittedly, many industrial accidents can be avoided by adequate guarding and fencing of the machinery as outlined in the factories acts, but it is the considered opinion of experts that certain standards of heating, lighting, ventilation, washing and sanitary arrangements, mess-room facilities and such like are essential requirements of a modern factory, and these can best be supervised by the personnel department in close co-operation with the medical services. Again, it is essential that in certain hazardous industries periodic medical examination of the workers should be carried out to detect early cases of health deterioration,—*e.g.*, lead-workers, or those handling commercial solvents such as carbon disulphide, trichlorethylene, chlorobenzene etc. and, if necessary, to transfer the worker from toxic to an innocuous job, for a time being, to enable him to recover before permanent damage is done to his health.

Welfare of the industrial worker can indeed be termed "a new discipline", leading to national economic stability. But it should be realised, of course, that industrial welfare is not the responsibility of any single department. It requires the combined efforts of the employer, the engineer, the chemist, the physician, and co-operation from the worker—all pooling their knowledge and experience for the promotion of a healthy environment in which to work. They must be aided and guided by a Central Industrial Health Research Institute, on the same lines as the Industrial Health Research Board of the Medical Research Council in England, whose function would be to collect and analyse all data from the country as a whole and from abroad. The Institute must be prepared to research under experimental conditions, to investigate and explore means of improving working conditions and environment, and act as an advisory body both on behalf of individual industry and the State. The employer, whether this be the State or private enterprise, must be enforced to adopt useful results of research and valuable recommendations by the Institute either by legislation or by public opinion. It is only through conscious teamwork between the State, the employer, and the worker, with a common research institute to help them, that production can be on the up-grade, industries may prosper and the Indian people attain and maintain a decent standard of living.

Conclusion

The object of this paper is to emphasize the need for an Industrial Health Research Institute at the centre, and promotion of industrial medicine alongside economic reconstruction and industrial development of the country.

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II

RESUSCITATION OF AYURVEDA

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India is now facing a great upheaval. Upheaval not only politically but also in every phase of science, art and culture. The problem of the health in the country also comes to the forefront. In these days of constituent assembly, national planning, post-war reconstruction etc., not only the state but also all right-thinking persons are paying close attention to the health problem in India. We have got before us four such documents dealing with this problem. These are —(1) Report of the Health Survey and Development Committee appointed by the Central Government with Sir Joseph Bhore as the chairman. This report is also called the Bhore Committee Report. (2) A thoughtful article, from the Prof. Sigerist, Director of the Institute of History of Medicine, John's Hopkins University entitled "The Need for an Institute of the History of Medicine in India" and published in the Bhore Committee Report (Vol. III, pp. 204-213) and portion of which (2 sections out of 5) has been republished in the Journal of the Indian Medical Association (**15**:390, 1946). (3) A special article "The Position of Medical Science in India" by Lt.-Col. G. R. McRobert published in the Indian Medical Gazette, (**79**:607, 1944) and quoted in the Committee Report. (4) The annual oration by Rao Bahadur Dr. B. Tirumal Rao on "On Some Aspects of Medical Education" delivered at the Annual Meeting, Vizagapatam Branch of the Indian Medical Association on 24th June 1946, and published in the Journal of the Indian Medical Association (**16**:13, 1946).

While referring to these four recent documents in the lines which follow, we shall refer them as (1) the Committee, (2) the Professor, (3) the Lt.-Col. and (4) the Rao Bahadur respectively. These documents give us various aspects of the health problem but in the following lines we shall restrict ourselves to the study of the problem dealing with indigenous systems of medicine only.

The recent (12-10-46) conference of the Health Ministers of provinces under the auspices of the Central Government having adopted the following resolutions, has given a great impetus to the advancement of indigenous systems of medicine. The conference resolved that in accordance with the recommendations of the National Planning Committee, adequate provision should be made at the Centre and the Provinces for research in and the application of scientific methods for the investigation of the indigenous systems like Ayurveda and Unani with reference to maintenance of health and prevention and cure of diseases; for starting schools and colleges for training for diploma and degree courses in indigenous systems of medicine; and for post-graduate courses in Indian Medicine for graduates in Western Medicine.

It appears that the above resolution adopted by the Health Ministers' Conference differs essentially from the recommendation as maintained in the above

critical junction in the history of India, any scheme relating to the medical relief and public health must be well planned and executed efficiently, one must study the relevant documents closely and critically.

The Indigenous Systems

It appears that in all the above documents including that of the Health Ministers' resolution the term indigenous systems of medicine has been very loosely used. By this term all have included Ayurveda and Unani to which the Rao Bahadur has added Homeopathy, Naturopathy etc. The dictionary meaning of "Unani" is "Greek". So Unani system means Greek system of medicine and it is actually so. It is not indigenous to India. It is rather Arabian Medicine based primarily on the Greek medicine incorporating Ayurveda to a considerable extent. The professor being a historian rightly says—"Unani Medicine, on the other hand, through the Persian and Arabic tradition goes back to Galenic, Hippocratic and Pythogorean views and having passed through the filter of Avicenna, it acquired many elements of Aristotelian philosophy."

From this point of view, by the term "indigenous system" we shall mean only the Ayurveda, with which we may include the Siddha System of South India, which although it has incorporated great amount of Ayurvedic doctrine, still had a separate origin of its own.

The Status of the Framers of the above Documents

It is a pity that persons who know little of a subject often think themselves to be experts in that subject. Such has, unfortunately, been the cases here. The Committee admitted that "We are unfortunately not in a position to assess the real value of these systems of medical treatment as practised today as we have been unable, with the time and opportunities at our disposal, to conduct such an investigation into this problem as would justify clear-cut recommendations. We do not, therefore, propose to venture into any discussion in regard to the place of these systems in organised state medical relief in this country." (Vol. II, p. 455). Yet they could not desist from the temptation of making recommendation.

The Professor undoubtedly a master of History of Medicine, reveals in his writing an unacquaintance with the science and practice of Ayurveda. Same with the Lt.-Col. and it appears that Rao Bahadur is innocent of the history as well as science and art of Ayurveda. It appears, further, that his inspirations, ideas, arguments and even the language, verbatim in places, are from the Professor.

We do not pretend to have a thorough and complete knowledge of Ayurveda, but from the little first hand knowledge of Ayurveda, we possess, we are in a position to say without any fear of contradiction that what has been said in these documents about the science of Ayurveda is not warranted.

Ayurveda *versus* Scientific Medicine

The Committee remark—"it has to be recognised that great improvements have taken place in the field of public health as the result of the many discoveries of science which are and can be implanted only through the scientific system of medicine and through personnel trained in such a system." (Vol. II, p. 455).

The Professor remarks—"At first sight it seems strange that these ancient indigenous systems are able to compete with modern scientific medicine." "Scientifically trained doctors will gradually replace the indigenous practitioners." "This would permit us to preserve and to incorporate into our scientific system of medicine whatever is found to be of value in the indigenous systems."

The Lt.-Col. remarks—"It means systems of medicine practised according to the ideas of hoary sages of pre-scientific Vedic times using only the means and methods known to the ancients with all their deficiences and drawbacks. By all means investigate any points of apparent value in these systems, and if suitable incorporate the facts into the general body of scientific knowledge."

The Rao Bahadur remarks—"He practises a system of medicine based upon concepts of anatomy, physiology and pathology which were conceived centuries ago and whose reality has come to be questioned at the hands of modern science."

All these remarks are undoubtedly not based on facts. We shall discuss presently the concepts of anatomy, physiology and pathology which were undoubtedly conceived about 4500 years from today but still they can stand the scrutiny at the hands of modern science. It is quite natural that Ayurveda is far backward when compared with the modern science only because thousands of research workers all over the world being patronised by the State and other bodies, and spending millions and millions of rupees during the hundred years are contributing to the modern science while Ayurveda has been really static, though temporarily, for the last several centuries. Excepting this period it was never static and has actually been the international scientific medicine being the mother of all medical systems of the world.

The editorial comment on the Lt.-Col.'s article (July-Dec. 1944) may be recalled here in this connection with the only addition that the Egyptian and Greek medicines also had been greatly influenced by the Indian medicine (*cf.* Antiquity of Hindu and Greek Medicine—by Dr. D. N. Banerjee, Calcutta, 1941). The editorial comment runs thus—"Scientific medicine is international and is not confined by geographic or any other boundaries. There was a time when in India medicine was probably as much or more advanced than in any country in the world. Indian medicine markedly influenced Chinese medicine, Arabic medicine and through the Arabians, European medicine, for even then medicine was already international."

Opinions Unwarranted

The opinions given in the above documents are now discussed.

1. Both the Professor and the Rao Bahadur have used the same language while speaking about the Tridosa doctrine, they say "(It is) an attempt to visualise the organism as a microcosm, and to place it in relationship with the macrocosm of the universe."

Both of them also say "Ayurveda is mainly philosophic in its outlook."

These words apparently originating from the Professor have been responsible for grave injustice both to Ayurveda and the Professor himself as these words have actually been misinterpreted, apparently by the Rao Bahadur. The proper significance and interpretation of the above statement becomes clear when read along with the statement of the same professor appearing elsewhere in the same document (Bhore Committee Report Vol. III, p. 212) which reads thus "The scientific medicine has a philosophy also. We took look at the human body as a microcosm in the midst of macrocosm (printed microcosm). The same elements that constitute the organism are found in the outside world and the same physico-chemical forces are acting in both."

Even a casual study of Tridosa doctrine (Tridhatu Siddhanta) studied from the angle of vision of modern physiology, will convince the reader, what a great amount of modern physiology (not philosophy) has been included therein. We shall refer to this matter while discussing on physiology of Ayurveda. Not only physiology, the pathology and therapeutics also are based on the Tridosa doctrine.

2. The Professor remarks—"The concepts of wind, bile and phlegm represented useful working hypothesis at their time, but the new science has demonstrated that the actions attributed to them are the result of speculation and do not correspond to a reality." The Rao Bahadur remarks simply "its physiology and pathology mainly speculative in conception." He however does not give any reason for the remark.

It is evident from his own remarks that the professor gets his experience from an English translation of Caraka Samhita and Susruta Samhita. It has long been recognised that the translators of Ayurvedic literature have made a great disservice to Ayurveda by translating Vayu, Pitta and Kapha as wind, bile and phlegm respectively. Vayu of Ayurveda is not wind, Pitta is never the bile as we find in the gall bladder, and Kapha is never the phlegm. Susruta says, the word "Vayu" is derived from the root "Va" meaning motion, "Pitta" from the root "tapa" meaning heat and "Shleshma" (the other name for Kapha) from the root 'shlesha" to unite. (Susruta—Sutra—21/5). It is natural for one unacquainted with Sanskrit to fall in the trap of wind-bile-phlegm translation and will come to the conclusion that it is speculation when he attempts to correlate how the manifestations attribute to Vayu, Pitta and Kapha in Ayurveda could be explained as the function of wind, bile and phlegm. It is not their fault, the fault lies with the translator. It is due to the same reason we find the remarks made by the professor. "The three elements, wind, bile and phlegm are symbols of the aerial, fiery and liquid forces of life-energy. Throughout its history Ayurvedic medicine was an attempt to interpret the phenomena of life and death, health and disease, philosophically." Ayurvedists, however, know that Vayu, Pitta and Kapha are definitely neither forces nor energy, they are all material substances.

Anatomy, Physiology & Pathology—the Basic Sciences in Ayurveda

Now about anatomy in Ayurveda. The Rao Bahadur remarks,—"The study of anatomy, physiology and pathology with which the student has started, can lead him logically only to the study of modern scientific medicine and not to any other system either ancient or modern." It is true that the knowledge of anatomy displayed in modern text books are not found in Ayurveda and further the modern Ayurvedists cannot explain even the amount of anatomical knowledge already extant in Ayurveda only because they have not dissected a human body during the last, say, 2000 years and that lack of knowledge had been responsible for interpolation of materials absolutely speculative. In spite of this, a keen modern anatomist will find abundance of material which will cause him a great surprise. Such had been to Hoernle when he made a keen study of Ayurvedic anatomy. (Hoernle, A. F. R.—studies in the medicine of ancient India, Oxford 1907, Part I. Osteology). Hoernle remarks —"Probably it will come as a surprise to many, as it did to myself, to discover the amount of anatomical knowledge which is disclosed in the works in the earliest medical writers of India. Its extent and accuracy are surprising, when we allow for their early age—probably the sixth century before Christ and their peculiar methods of definition."

A modern anatomist with knowledge in Sanskrit, will undoubtedly profit much if attention is directed to this in research spirit. One orthodox Ayurvedist with no knowledge of dissection will be of no help as it will not be possible for him to decipher their peculiar methods of definition referred to by Hoernle.

Further, both major and minor surgical operations, such as laparotomy, operations on the intestines, Cæsarean section, embryotomy, lithotomy, amputation of limbs, and various plastic operations described by Susruta and Vagbhata, and also the operation on the brain of King Bhoja as described in Bhoja Probandha, force us to conclude that the great precision with which the above operations are described presupposes that the anatomical knowledge could not have been of a mean order.

It is also surprising how the ancient sages could visualise the unseen, we mean, the microscopical anatomy. One passage from Caraka is very significant. Caraka says—"The component elements composing the body are innumerable when considered in terms of *paramanus* (finest units) as they are extremely large in number, very fine, in fact, beyond the conception of all senses." (Caraka, Sharirasthan—7/12). When considered in terms of modern science, these innumerable invisible elements composing the body come to indicate the cells.

Physiology in Ayurveda

The Tridosa doctrine represents the physiology in Ayurveda. Vayu, Pitta and Kapha are the three main principles called the three dosas (Tridosas). We must first remind the reader that these three dosas, according to Ayurveda, are material substances, having

weight. They are constantly being produced and so increase in amount and also constantly being depleted. They are not energies and possess definite qualities.

Broadly speaking, one may say that many functions of Vayu are performed through the nervous system hence this system may be termed the Vayu apparatus. Pitta represents the enzymes and hormones and regulates metabolism. Kapha refers to composing elements of the cells of the body and also the body water. Here we refer only a few salient features on Vayu. Banerjee (1945) has pointed out that the Samhitas present us with at least 141 functions of Vayu when in normal condition and 821 symptoms being produced by Vayu when vitiated. It is remarkable that all these 962 manifestations of Vayu can be most appropriately classified in terms of modern physiology as follows—(1) Symptoms due to normal and abnormal mental functions; (2) those of special sense organs; (3) those of cardiac action, respiration, deglutition; (4) those of speech; (5) those of digestion, (6) of excretion, (7) of vasomotor system; (8) motor function; (9) sensory function and (10) general functions. It is evident that all the above functions and symptoms produced by Vayu represent the functions of the nervous system. In terms of Ayurveda, Vayu is divided into five groups called Prana, Udana, Samana, Apana and Vyana. Reclassifying the above manifestations of Vayu under these 5 groups it is found that the Prana Vayu is responsible for first 3 groups of the above classification, Udana Vayu performs the function of speech, Samana Vayu of digestion, Apana Vayu of excretion and parturition and Vyana Vayu of the next 3 groups. The last group—general functions refer to those of each individual cell of the body. It is surprising how the functions of Vayu apparatus (the nervous system) have been adequately described 4500 years ago. Apart from the functions attributed to Vayu, the function attributed to hrdaya (meaning both the heart and the brain), dhamani (nerve) etc. show surprising knowledge of the nervous system. To mention one item, our knowledge on autonomic nervous system dates from the works of Langley who published his book in 1921. It is amazing to find the description of the function of Prana Vayu as regulating circulation and respiration (group 3 of above classification), Samana Vayu in regulating digestion (group 5), Apana Vayu in regulating excretion and parturition (group 6) and Vyana Vayu in regulating the vasomotor function. The observation in Ayurveda are so well up to the mark that when one compares the function of, say, the Apana Vayu or Samana Vayu of Ayurveda with those of the inferior mesentric ganglia or the cell stations in the collateral ganglia of the solar plexus, the semilunar and superior mesenteric ganglia as described in any modern text book of physiology he may be tempted to remark that one is the translation of the other. This comparison is given below—(A) Comparison of the seat and function of Apana Vayu with those of the inferior mesenteric ganglia—Caraka says,—The Apana Vayu throws out all impurities and gives form to the embryo. (C.S. (12/8). He further says, "the testis, the pubic region and the bladder, the male organ, the naval, the thighs, groins and anal canal are the seats of Apana Vayu. Seated in the pelvic cavity it expels the semen, the urine and the stools. It is this Vayu which also expels menstrual blood and the fetus." (C.Ch. 28/9). Susruta also says, "Apana Vayu is seated in the colon. Its functions consist in bearing down the fetus and the faeces and in evacuating the urine, semen and menstrual blood." (S.N. 1/11).

Comparing these with modern science we find in Starling's Principles of Human Physiology—5th. Ed. 1930, p. 374, while indicating the main course and functions of the fibres of the sympathetic system—"The pelvic viscera are supplied by the lower dorsal and upper three or four lumbar nerve roots. These fibres also pass by the main chain to make connections with the cells, chiefly in the inferior mesenteric ganglia. They convey—vaso-constrictor impulses to pelvic viscera, inhibitory fibres to colon (both coats), motor and also inhibitory fibres to bladder, motor fibres to retractor penis, motor and inhibitory fibres to uterus and vagina."

About Samana Vayu we find in Ayurveda—

Susruta says,—"The Samana Vayu courses along the wall of the stomach and the intestines. Its functions consist in digesting the chyme brought down in the intestines in union with the digestive fire (ferments) and specially in disintegrating its essence from its refuge (*i.e.*, the matter to be excreted)."—S.N. 1/9.

We find in Starling's Physiology—

"The abdominal viscera receive fibres from the lower six dorsal nerves and the upper three or four lumbar. Most of these fibres run through the sympathetic chain and have their cell stations in the collateral ganglia of the solar plexus, the semilunar and the superior mesenteric ganglia. Their functions are—vaso-constrictor for stomach and small intestine, kidney and spleen. Probably vaso-dilator for the same viscera. Inhibitory for both muscular coats of stomach and small intestine. Motor for ileocolic sphincter, secretory to the suprarenal medulla. Vascular and glycogenolytic to the liver."

The very terms solar plexus and semilunar ganglia are also of great interest when we compare the seat of Samana Vayu as mentioned by Bhavamisra (13th century A.D.) quoting from a previous author, thus—the lunar plexus (सोमस्य मन्डलम्) is situated at a level a little to the left side of the naval. Inside the lunar plexus is situated the solar plexus (सार्यमन्डलम्) and inside the solar plexus is the seat of agni (digestive fire)—(Bhavaprakasa—Purva—Part I).

Similar knowledge of the ancient Hindus are found with many other aspects. Of course, we agree with the Rao Bahadur when he says, "there was very little apparatus to aid their observation."

The tragedy is that the orthodox Ayurvedist cannot appreciate the explanation of Ayurvedic doctrine in terms of modern physiology while the modern physiologist has got no first hand knowledge of Ayurveda.

Pathology in Ayurveda

Etiology in Ayurveda divides itself into two categories—direct and indirect. Direct causes—All the diseases are produced directly as a result of vitiation of the Tridosa—Vayu, Pitta and Kapha. The different diseases are produced according to (*a*) which of the dosas is or are predominating and (*b*) which tissue or organ has been affected. The pathogenesis of the disease are discussed under the term Samprapti. The fundamentals of Ayurvedic pathology is thus based on the Tridosa and so mainly deals with how the dosas cause derangement of the soil, the body.

Indirect causes—These are the external factors. The external factors are threefold—faulty food, faulty ways of life and the season. These external factors are responsible for causing derangement of the dosas, the direct cause of disease. Unless and until the dosas are deranged the external factors cannot cause disease in spite of their presence.

Microbiology in Ayurveda—It may appear ludicrous to conceive of any evidence of microbiology in Ayurveda only because of the fact that no one anywhere in the world can think of microbe before the invention of microscope. Leeuwenhoek discovered protozoa in 1675 and bacteria in 1687. That microbe is the cause of disease came to be known in 1845. From this we can infer that no one could think of pathogenic microbe before 1945. It is amazing to find the knowledge of pathogenic microbe in Ayurveda and other ancient treatises. They are generally termed as invisible krimi (worm) or invisible raksha, pishacha etc. There are mention of these in Caraka and Susruta Samhitas in the causation of ulcers, pus formation, different kinds of diseases of children. Apart from this Atharva Veda mentions of Takman, the causative organism of malaria. Yogbashista Ramayan mentions how the huge rakshashi named Karkati gradually became smaller and smaller ultimately invisible but of the form of a needle and thus got the name Visuchika (cholera) and ordained to remain in filth and to attack human being causing Visuchika (cholera)—(Utpatti prakaran—Ch. 68-84). The whole description agrees essentially with the etiology of cholera.

Infection and infectious disease in Ayurveda—Modern historians say that Fracastoro (1546) was the first to think of contagion and contagious diseases. We may quote a passage from Susruta (S.N. 5/26) which gives a definite idea of the knowledge of this condition. He says "coitus, contact with skin and breath, taking meal from same receptacle, sleeping or sitting on the same bed, using the cloth, garland and cosmetic used by a patient, transmit skin diseases, fever, consumption, conjunctivitis, and infectious diseases from one person to another." This teaching of Susruta holds good to this day. There is no space here to deal with even a very brief summary of the vast material as regards pathology and clinical medicine dealt with in Ayurveda.

A very casual study of Ayurvedic pathology (Nidana) will convince the Rao Bahadur that he is not justified in remarking that "diseases were studied mainly by observation of a few symptoms and signs depending mainly on mother wit and shrewd intuition."

Medicine and Therapeutics in Ayurveda

The committee remark—"We realise the hold that these systems exercise not nearly over the illiterate masses but over considerable sections of the intelligentia." The professor remarks—"The fact that indigenous medicine plays such a very important part in the life of the Indian people is frequently minimised or even ignored—its popularity is growing rather than diminishing." The Rao Bahadur remarks—"These systems of medicine still greatly appeal to the imagination of even our educated public."

One must also attempt to visualize the reason of popularity in the selection of the profession also. The Rao Bahadur informs us, "It is computed that there are at least 40,000 practitioners of Ayurveda in the province of Madras itself." While according to the committee Report there are 47,400 doctors in the whole of British India (Vol. II, p. 58). This shows that the Ayurvedic practitioners all over India constitute several times the numbers of doctors even after more than a century of establishment of medical colleges in India. In big cities the Ayurvedists often command bigger fees than the best doctor and they compete very efficiently with the best specialist having full laboratory facilities.

The Committee have made a great injustice in making the recommendation and planning taking only the 47,400 doctors as the only practitioners, absolutely ignoring the ayurvedic practitioners who are many times this number and competing efficiently with the general run of doctors.

The finding of the Lt.-Col. is significant. He says, "It is most disconcerting to find that at this late date not only are the hundreds of millions of the ignorant peasant population pathetically contented with indigenous systems but that a high proportion of the educated classes are of like mind. I have a number of Indian friends and acquaintances on the bench of the High Courts of judicature and among senior Indian counsel. I have been disappointed to find that nearly all have a strong predilection for the indigenous systems."

The reason is obvious. The methods of diagnosis and treatment still extant in Ayurveda are quite competent to impress them about their efficiency.

Public Health and Ayurveda

The Committee observe, "Thus public health or preventive medicine, which must play an essential part in the future of medical organisation is not within the purview of the indigenous systems of medical treatment as they obtain at present." (Vol. II, p. 455). This statement is undoubtedly true when read with "as they obtain at present." Otherwise it is far from the truth. Just compare with what Garrison (History of Medicine—p. 63) and Castellani and Chalmers (Tropical Medicine) say about this point.

Garrison says "Witness the Susruta's very recognisable description of malarial fever, which is attributed to mosquitos or the passage in the Bhagavata Purana which warns people to desert their houses" when rats fall from the roofs above, jump about and die" pre-

sumably from plague. Evidences of variolation (inoculation against small-pox) have been found in the Sanskrit text Sacteya, attributed to Dhanwantari (p. 63). He says again, "Human inoculation of variolous virus is said to be mentioned in the Atharva Veda" (p. 387).

Castellani and Chalmers observe "There is no doubt that the Indian doctors were well versed not merely in medicine and surgery but also in the prevention of disease and operative midwifery. Among the various remedies must be mentioned the fact that inoculation of small-pox was practised at the beginning of the warm season. Dietetics and toxicology were also well-known. Hospitals were founded by Buddhist princes in India and Ceylon. In fact, there is an account of a hospital being founded in Anuradhapura, the ancient capital of Ceylon, as early as the 5th century B.C. and later many more were established, as well as a sort of medical or sanitary department, having one medical officer to every ten villages, together with a definite sanitary organisation, and with institutions for the reception of cripples, deformed and poor persons."

We commend this last sentence to the attention of the members of the Committee. How many institutions for the reception of cripples, deformed and poor persons are there in the whole of India at the present moment? There had been sanitary department having one medical officer to every ten villages in 5th century B.C. How many are there at the present moment—25 centuries after the achievement of ancient India? The Committee have given the figures (Vol. I, p. 47, para 18).

"In 1935 nearly half the districts and three quarters of the municipalities in British India had no qualified health officers. Between 1935 and 1944, the number of whole time health officers, who are graduates in medicine and possess a public health qualification, increased only by 6 and the corresponding number of health officers who are licentiates increased by 18. During the same period the number of sanitary or health inspectors employed in the country as a whole increased by 656, numbering a total of 2976."

The Committee report further, "Even in those provinces in which the public health organisation has been best developed, *e.g.*, the provinces of Madras and the United provinces, the strength of the staff available is quite inadequate for the large territories and populations entrusted to them. In the province of Madras, for a district with an average area of 5,256 square miles and an average population of over two million, the staff consists of one District Health Officer and in addition of one Asstt. Dist. Health Officer in most districts with a Health Inspector in each taluk." (Vol. II, p. 139). Compare this with the time of King Asoka when there was one medical officer to every ten villages.

India *vs.* America, Europe and Africa

The Professor has said, "India is neither Britain or America". We may add Africa to that from the point of view of preventive medicine and that with respect to three preventible diseases, malaria, small-pox and cholera.

Malaria—The Committee mention—"At least 100 million persons suffer from malaria every year, and the annual mortality for which the disease is responsible, either directly or indirectly, is about 2 million (Vol. IV, pp. 7-8). A tragic feature of the situation is that much of the malaria in the country is man made. In many cases roads, railways, and irrigation projects have a sinister account to their credit" (Vol. IV, p. 34). "We should refer to the estimate made by Lt.-Col. J. A. Sinton, who places the annual loss to India on account of malaria alone any where between 147 and 187 crores of rupees." (Vol. II, p. 35).

Such is the state of affairs in India 49 years after the discovery by Ross (1897) of anopheles as the vector for malaria. Compare this with America. Reed (1901) discovered that the virus of yellow fever is transmitted by a particular species of mosquito, the Stegomyia fasciata. Garrison (p. 752) informs us: "In February 1901, shortly after Reed had proved his case, Gorgas, as chief sanitary officer of Ravana, Cuba, began to screen yellow fever patients and destroy mosquitos, and in three months, Ravana was freed from the disease for the first time in 150 years. In connection with the work on the Panama Canal, Colonel Gorgas has freed that part of the isthmus not only from yellow fever, but from all dangerous infections, and through this great triumph in sanitation, Panama formerly a notorious plague spot of disease, the "White Man's Grave", as it was called, is now one of the healthiest communities in existence. And this had been achieved in course of one year and a half. India is certainly not America!

Cholera—An epidemic of cholera was reigning in Egypt in 1883. Robert Koch was sent by the German Commission to investigate the etiological factor result ing the discovery of the cholera vibrio (1883). There had been only two epidemics of cholera in Egypt since then, in the years 1895 and 1902. Egypt has been able to rid herself from cholera within such a short period of only 20 years. While in India about 300,000 people die of cholera every year. India is not Egypt even.

Small-pox—The International Sanitary Convention by regulating international traffic by sea and air have made the prevalence of small-pox in European countries a thing of the past. The Bhore Committee inform us, "The success that can be achieved in eliminating small-pox through vaccination has been demonstrated by the history of the disease in the Philippine Islands. For many years small-pox caused more than 44,000 deaths annually in that country. After the introduction of systematic vaccinations the disease almost disappeared" (Vol. II, pp. 168-9). So far as India is concerned, the Committee report, "The annual epidemiological reports published by the League of Nations show that India has the highest rate of incidence of small-pox among the countries for which statistics are given." (Vol. II, p. 167). India is thus not even like the Philippine Islands!

This is the state of affairs in India with the State solely in charge of the public health problems.

It is useless to acuse that the public health or preventive medicine is not within the purview of the indigenous systems of medical treatment." (Vol. II, p. 455).

RECOMMENDATIONS

The Rao Bahadur recommends, "I believe there is still a great deal to learn from the ancient systems of Medicine. They have got an extensive and wonderful pharmacopœia. They have developed dietetics almost to a fine art. These systems require to be studied therefore in all their several aspects and all that is useful and valuable in them can be easily absorbed and incorporated into the modern medicine."

The Col. recommends, "By all means investigate any point of apparent value in these systems and if suitable incorporate the facts into the general body of scientific knowledge (p. 611).

The Professor recommends, "The Institute (of the History of Medicine in India) will therefore investigate the indigenous medical systems, not only for their ideological content, not only as aspects in India's ancient and mediæval civilizations and as end product of a long development; it will also endeavour to evaluate their practical achievements. This would permit us to preserve and to incorporate into our scientific system of medicine whatever is found to be of value in the indigenous systems. And it would also permit the objective refutation of claims that are not justified." (Bhore Committee Report Vol. III, p. 211).

Lastly the Committee recommend, "We feel that it would be unfair and unjust merely because some other method of treatment is said to be cheaper, to deny to any one in this country the benefit of the scientific system and of the daily volume of research and achievement in the wide world of science. We feel that it should be left to the Provincial Governments to decide what part, if any, should be played by the indigenous systems in the organisation of Public Health and Medical Relief." (Vol. II, p. 456).

From these recommendations it appears that, excepting the Committee who have left the decision to the Provincial Governments, all the other three are of the impression that Ayurveda possesses rather mainly "an extensive and wonderful pharmacopœia" and "to incorporate into our scientific system of medicine whatever is found to be of value in the indigenous systems." This is only because these scientists have come into fragmentary contact with the therapeutical aspect of Ayurveda in their practice while they are absolutely innocent of the other branches of the science of Ayurveda.

We believe that Ayurveda may also reciprocate the same as recommended by the learned authors under review. Ayurveda may also absorb and incorporate the fundamental sciences of physics, chemistry and microscopy which have firmly established the modern science, and these will also glorify Ayurveda as they have done the modern science. The part played by them in this achievement has been very adequately described by the Col. and also quoted by the Committee. (Vol. II, p. 456). The description runs thus:—

"It was not until the middle of the 19th century that medical science became firmly established on a secure foundation. The invention of the compound microscope, the rapid development of Organic Chemistry and latterly of Bio-Chemistry and Bio-Physics have led to such an advance that we can say with truth that 95 per cent of the total corpus of knowledge with regard to the working of the human body has been obtained within the life time of men who are still with us. It may surprise some to know that nearly all advances in the science of medicine now come from men who have nothing to do with medical practice or with the care of the sick but who have a special knowledge of and training in the fundamental sciences of Physics, Chemistry and Physiology."

Nearly all the learned writers under review have mentioned that Ayurveda is static and the Rao Bahadur remarks,—"There are those who claim that Ayurveda is self-contained and self-sufficient according to its own postulates of physiology and pathology and is capable of being worked as an effective system of medicine. It does not admit of any additions or improvements without violating its basic principles and theory." This is undoubtedly the common belief but that is far from the truth. The best armamentarium of the present day Ayurvedists in diagnosis is the pulse lore and in therapeutics, preparation containing mercury and sulphur called the Rasa preparations. Rasa here means mercury. It appears that the pulse lore and Rasa preparations were unknown to Agnivesa (C. 2500 B.C.) (the writer of the Samhita now known as Caraka Samhita), Susruta (2100 B.C.). Caraka (1st century A.D.), Vagbhat (6th century A.D.), Madhabakara, author of Nidana (7th century A.D.), Cakrapani (1060 A.D.) even up to 15th century A.D. We find mention of the pulse lore for the first time by Bhabamisra (16th century A.D.) and early mention of Rasa preparation by Chakrapani (1060 A.D.). The pulse lore and the Rasa preparations are the contributions of the tantric scholars, foremost being Adinath, Nityanath etc., who circulated their knowledge in about 12th or 13th century A.D. The Ayurveda had not been slow to incorporate them. Ayurveda today will also not be slow to incorporate the basic principles of modern science.

CONCLUSION

In conclusion, from what we have said about Ayurveda, both past and present, we feel to concur with Dr. A. H. Butt, Director of Public Health, Punjab; Dr. U. B. Narayan Rao, President, All-India Medical Licentiates Association; and Dr. Vishwa Nath, Member, Medical Council of India the three members of the Bhore Committee who made a more positive recommendation which runs as follows:—

"We are of the opinion that the teaching of indigenous systems of medicine should be regulated by the State. The Bombay Medical Practitioners Act, 1938, represents in regard to registration, the medical curriculum and examinations preliminary to registration, a step in the right direction. Practitioners trained and registered under the requirements of the above Act, or similar legislation, should be freely utilised for

promoting public health and medical relief in India." (Committee Report. Vol. II, p. 461).

The resolution, adopted by the Health Ministers' Conference (quoted at the beginning of this review) about adequate provision for research in and the application of scientific methods for the investigation of the indigenous systems of medicine, is the most appropriate and right approach to the problems of medical education and maintenance of health and prevention and cure of disease.

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(*Concluded from page 122*)

Delayed grafts—Brooks and Hudson have developed another method of bone transplantation in experimental animals and Key has adapted the method to operation on human beings. In the first stage the transplant is cut on the donor at the site but not removed. In the second stage, several days later, the transplant is removed and placed in its new bed. The biologic explanation of this fact is simple. The preliminary preparation leads to a rarefaction of the bone and consequently augments the permeability of the passage for rehabilitation.*

* Read at the Scientific Section of the XI U.P. Provincial Medical Conference, Saharanpur, October, 1945.

(*Concluded from page 132*)

This treatment continued for 84 hours after which Penicillin was obtained. Improvement so far noticed was a rise in temperature to 99·4°F., urination and defæcation, a slight lessening of the retraction, and he could be fed just a little.

Penicillin was started at 2 A.M. intramuscularly every three hours in doses of 20,000 units. On the same day at 1 P.M. when I saw him his condition was appreciably better. Temperature 99·8°F.; pulse 124, good volume and tension and regular; respiration 12; consciousness seemed to be coming back; when the spoon was held before his face he opened his mouth and swallowed all right. I was told that he had been taking a lot of fluid since morning.

Lumbar puncture was allowed, and at 2 P.M. after withdrawing 10 c.c. of almost clear fluid which ran in rapid drops, 10,000 units of penicillin in 10 c.c. double-distilled water was injected intrathecally. Intramuscular penicillin continued as before.

At 10 P.M. temperature was 101°F. and the patient was sleeping quietly. Suddenly it was found that his head and lower extremities were being drawn back and the body became stiff. The extremities gradually relaxed but the head remained retracted and stiff and there was also lock-jaw. The patient lost all consciousness. Several fits affecting the legs and head occurred in succession and the temperature began to rise; by 5 A.M. it was 105·4°F. No more fits followed. Temperature gradually came down but the condition grew worse till the child died at 10-30 A.M.

Recrudescence and relapse are common in meningitis. The epileptiform convulsion and fever suggest cerebral irritation due to penicillin or meningoencephalitis—ED., *J. Indian M. A.*

(*Continued from page 142*)

types) is the infective agent. Sore throats or upeer respiratory infections commonly precede the rhematic attack, and epidemics of streptococcic sore throat have often been accompanied or followed by epidemics of rheumatic fever with carditis in a proportion of cases. Serologic reactions in rheumatic children support the theory of an initiating steptococcic infection. There is growing evidence of the efficacy of sulphonamides when used prophylactically to prevent rheumatic recurrence, though they have no beneficial effect on the rheumatic attack.

There is strong evidence that the rheumatic attack is a specific response at a susceptible age in individuals with an inborn or acquired sensitivity to a common agent. As with other sensitization phenomena, the rheumatic attack follows the throat infection after an interval up to two or three weeks and its articular and cutaneous manifestations, like those of serum sickness, are transitory. On the one hand the existence of rheumatic families, with the disease appearing in successive generations under different environments and, on the other hand, the escape of most children living under unfavourable conditions, suggest that at the two ends of the scale there are two types that are more or less susceptible to infection or more or less liable to develop a sensitized state in the presence of infection.

Social factors are important. Rheumatic fever and heart disease are more common in working class populations than among the well-to-do. Of the factors operating within the framework of poverty, damp houses have often been suspect, but crowding, with its increased liability to droplet infection, seems to be more definitely incriminated. If, as is now generally accepted, droplet infection with streptococcus is an important factor, outbreaks might be expected among young adults well cared for but subject to overcrowding. In fact, outbreaks have occurred in training ships and barracks where other influences were not unfavourable except damp and chill. But if these are potent factors why was rheumatic fever so rare in young soldiers during the trench warfare of 1914-1918? Social studies of individual cases in children will frequently reveal adverse conditions of life before the attack of rheumatic fever. The domestic outbreak of streptococcic infection is worthy of a much closer study.

CASE NOTES

A CASE OF DEXTROCARDIA

S. VENKATESWARA RAO, M.D. (ANDHRA),
Physician, S. B. Hospital,
Hyderabad-Dn.

Ahmadi Begum, female child of 10 years was admitted in my ward on 20-5-46 for irregular fever of 2 months duration.

On examination the child looked pale.

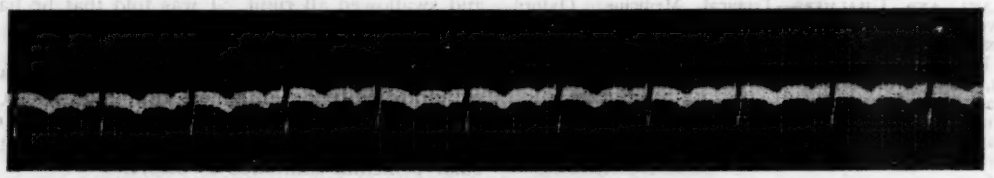
Cardiovascular system—the apex-beat was visible and palpable in the right fourth interspace just internal to the right midclavicular line. The pulse rate was 72, regular, of fair volume and tension. On percussion the upper border of the heart was found along the upper border of the third rib and the left border along the left lateral sternal line. Blood pressure was 110/70 mm. of Hg. Heart sounds were normal in all the areas.

Lungs—nothing abnormal detected.

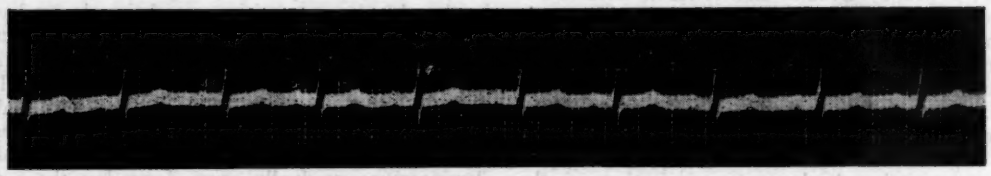
Abdomen—soft. Liver and spleen not palpable.

X-ray of the chest—dextrocardia of the heart noted. (see plate facing p. 113).

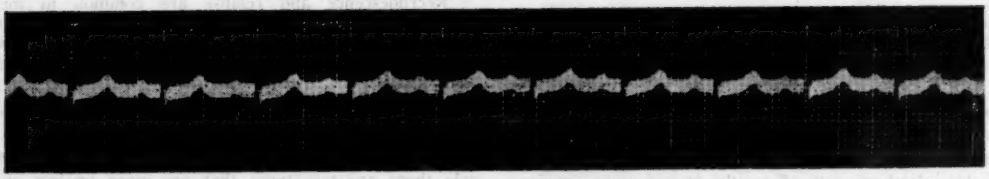
Electrocardiogram—"Rate 90 per minute. Evidence of dextrocardia—Note the inversion of P, R and T in lead 1.



LEAD NO. 1—BETWEEN RIGHT AND LEFT ARMS



LEAD NO. 2—BETWEEN RIGHT ARM AND LEFT LEG



LEAD NO. 3—BETWEEN LEFT ARM AND LEFT LEG

Acknowledgments—I wish to thank Raja Bahadur Lt.-Col. K. N. Waghray, M.B.B.S., M.R.C.P. (LOND.), N.M.S., Director, Medical and Public Health Department, for permission to publish this case; and Dr. Nasiruddin, Radiologist, Osmania Hospital, Hyderabad, for the skiagram and electrocardiograms.

The x-ray plate suggests transposition of viscera also along with the dextrocardia, an important clinical finding—ED., *J. Indian M. A.*

A FATAL CASE OF MENINGITIS

N. C. DAS GUPTA, M.B.
Khulna

Male child, 4 years old. On the 8th day of fever he became comatose and had stiffness of the neck. He had two injections of quinine on consecutive days early in the attack. I saw him on the 10th day and found him in deep coma with considerable retraction of the head, corneal and iris reflexes absent, pupils contracted, Kernig's and Brudzinski's signs present, knee jerk exaggerated; temperature subnormal from the previous day; pulse 160 per minute, thready, regular; bowels constipated, tongue thickly coated and dry, abdomen scaphoid, spleen and liver not palpable; no urine for over 24 hours, neither any accumulation in the bladder. There were occasional spasms of the left arm and leg. He had been taking no food, everything given by mouth was flowing out. He had only 1 gm. sulfapyridin intramuscularly.

He was immediately put on sulfathiozol 1 gm. intramuscularly every 4 hours, coramin, adrenalin and glucose parenterally and rectal saline. Penicillin was sent for. Lumbar puncture not permitted by parents.

(*Concluded in page 131, col. 2*)

JOURNAL OF THE

INDIAN MEDICAL ASSOCIATION

CALCUTTA, JANUARY, 1947

INDUSTRIAL HEALTH

Post-war India has been marked by large scale industrial labour unrest. This is not a peculiar feature of this country alone. Countries of the west are suffering from the same malady. Inflation, high prices of commodities combined with greater realisation of labour utility during World War II, have made such unrest inevitable. In our country there are some additional reasons which must be faced boldly. It cannot be denied that a majority of the millowners had been totally apathetic towards labour welfare. A large number of *'Badlis'* added to this unfortunate attitude. The Government passed some Labour Acts which were hardly enforced in factories. The Acts did not provide for any improvement in the living conditions, environments or health of the workers. Statistics of factories and reports of the mines give only a general figure of 'accidents'. No detailed analysis of the various general diseases or diseases peculiar to industries are mentioned nor has any enquiry been made yet. One appreciates that the migratory nature of workers makes this enquiry difficult. But it must not be forgotten that the prevalent practice of the employer is to dismiss a worker the moment he reports ill or unable to carry on the job. Crushed between the attitude of the millowners and the labour, the industry of the country suffers. It is time both the parties realised this untenable situation. Where they fail, Government should step in. The recent Health Insurance Scheme for Industrial Workers introduced in the Assembly is a step in the right direction.

Chakravorti[1] has lately drawn attention to this human problem of work. In another communication published elsewhere in this issue[2] he has elaborated his theme further. On the eve of large scale industrialisation in our country, these deliberations are none too soon. Western countries have advanced far in this direction and they learnt from their difficulties and failures. It is reasonable to expect that we should not wait till the difficulties arise but start planning from now and take advantage of the experience gained by the industrialists of other countries.

The problem of industrial health in India is not an unwieldy one. Comparatively only a small percentage of the whole population is engaged in the industry. It is estimated that only 20 million out of a population of 400 million, are dependent on the industry for their living. Only some 2½ million *i.e.*, 0·6 per cent of the population are actively engaged in regular industrial organisations. A tour along the so-called 'cooly lines' or labour quarters will convince every one about the miserable conditions of the workers. They are underfed, badly housed and the environments are filthy. Naturally they fall easy victims to various diseases. The industrialist finds it cheap to dismiss him and replace him by another man instead of investigating the cause of illness or trying to prevent such illness. This method is bringing about fresh conflict between the employer and the employee resulting in loss of working days. If many workers of the 'occupational age group' fall ill, they eventually become a burden on the country and no state can lose this huge 'man power hours'.

It is time when to take into consideration the diseases or accidents in a man from the machine or his occupation. It is a sad reflection on us that we have not yet come across cases of pneumonoconiosis or byssinosis in the reports of the mines. We know that a large number of people are employed in mines and cotton mills and many are working for a long time. There is no reason to believe that these people are immune to pneumonoconiosis. There is also no report on toxic hazards or other occupational risks. It only shows lack of any investigation in these directions. As there is no method of preemployment or periodic medical examination, the chances of detection of diseases are remote. It has been established that some people are physically and mentally unfit for c tain chemical and mining industries. It has also been revealed that some people are psychologically misfit in certain types of work. These are the workers who are just like 'a square peg in a round hole'; they are the victims of various types of industrial dermatitis, respiratory diseases and are 'accident prone'.

Shortage of skilled labour makes it a necessity that workers should be protected against industrial and general diseases. In the selection of personnel for any industry, the inclination of the person for the type of work should also be seriously considered. Failure to take such steps will act like a brake on the production. Man must be treated as a part of the machine. He must also be cared for like the machine. We mean that the health problems of the workers should be the concern of the industrialists or the state. The problem can be rightly tackled by the close collaboration of the employer, employee, engineers, chemists and the physician. Their work will be aided and guided by a Central Industrial Health Research Institute. Such an institute has been a great help to the industrialists and workers of Western countries. The Government should take immediate steps for the establishment of such an institute separately or as a part of the Central Medical Institute which the Government intends to start in near future. If the industry of the country is desired to progress without any set back, the state or employer must look upon the problem of industrial health from this perspective.

[1] CHAKRAVORTI, S.—*Indian M. Gaz.*, **81**:370, 1946.
[2] *Idem—J. Indian M. A.*, **16**:123, 1947.

CURRENT TOPICS

PALUDRINE AND MALARIA

Dr. B. B. Roy, M.B., (Cal.), from the Department of Pharmacology, Carmichael Medical College, Calcutta, gives in the following lines a comprehensive review on the subject (*Carmichael Med. College Mag.*, **15**:70, 1946):

Introductory

Paludrine was synthesized and first tested in avian malaria in 1945; in the same year it was also tried in the treatment of human malaria. The synthesis of paludrine has been the outcome of planned research and developed in logical steps. Curd, Davey and Rose[1] stated their work by investigating the activity of pyrimidine and its derivaties, not as is the case generally, with the basic structure of pamaquin (quinoline) or mepacrine (acridine). Pyrimidine was chosen for several reasons of which one of obvious interest was that certain sulpha drugs, sulphadiazine (sulphapyrimidine) in particular, possessed antimalarial activity. In the course of their investigation several compounds were obtained which showed some antimalarial activity. Planned research soon revealed that inclusion of guanidine group greatly enhanced antimalarial activity. The first compound (3349) proved effective in human malaria but was rejected for its unpleasant side effects. Derivatives of biguanidine were then prepared, leading ultimately to the synthesis of two potent products (4430 and 4888). The product 4888 was found to be superior to the other and virtually nontoxic; it was given the name, "paludrine". Paludrine is a colourless bitter compound, and chemically, it is N_1-p-chlorophenyl—N_5-isopropyl biguanidine.

Biology of the Malarial Parasite in the Vertebrate Host

The importance of the discovery of paludrine can only be fully appraised by an appreciation of the present-day conception of the biology of the malarial parasite in the vertebrate host, as has been studied in avian malaria. It is presumed that similar conditions are obtained in human malaria. The position may be briefly summarised after Davey[2] with respect to *P. gallinaceum* infections with which tests for activity of paludrine were conducted in birds.

When sporozoites are introduced into the blood stream they do not persist there for more than a short time but quickly penetrate the tissues. Within the tissue they pass through a development phase. The duration of this 'primary tissue phase' constitutes the incubation period of the infection. Following the end of developmental primary tissue phase there is a release of parasites into the blood stream and into further tissue cells, giving rise to a secondary tissue phase. The primary tissue phase is essentially developmental and only follows the introduction of sporozoites; this phase is probably common to all malarial parasites. The secondary tissue phase may be formed from the primary tissue phase or it may be initiated by blood parasites, as diagrammatically shown below:

Sporozites
↓
primary tissue phase ⟶ secondary tissue phase
↓ ↑ ↓
Blood phase

This secondary tissue phase has been definitely described only in case of *P. gallinaceum* and is perhaps a specialised feature of only a few malarial parasites. Unlike the primary tissue phase the secondary tissue phase is capable of indefinite schizogony, and may persist as long as infection lasts. A feature of these extra-erythrocytic parasites is their great refractoriness to drugs such as quinine and atebrin, and it is suggested that such forms may be present in human malaria, particularly in *P. vivax* infections, and constitute the explanation of why relapses so frequently occur.

The foregoing considerations have their bearing on the problems of malarial chemotherapy. There may be five forms of malarial parasites which react specifically to drugs:

(*i*) The sporozoites;
(*ii*) the primary tissue phase—the primary exo-erythrocytic forms;
(*iii*) the secondary tissue phase—the secondary or persistent exo-erythrocytic forms;
(*iv*) the schizonts of the red blood cells; and
(*v*) the gametocytes.

Causal Prophylaxis

A true causal prophylatic drug will act either on the sporozoites or on the primary tissue phase, but a drug which suppresses the blood phase as it arises from the primary tissue phase will stimulate casual prophylactic action (in fact clinical prophylaxis only) and this is what is obtained after prophylactic administration of a drug like mepacrine. True causal prophylaxis remained an unattainable ideal before the introduction of paludrine. "Paludrine has proved superior to all known antimalarial drugs, as in non-toxic doses it is a true causal prophylactic, exerting a powerful lethal effect on the exo-erythrocytic (presumptive exo-erythrocytic) forms of *P. falciparum* and fully protecting volunteers receiving viable sporozoites by mosquito inoculation. In *P. vivax* sporozoite-induced infections it is only a partial causal prophylactic; the exo-erythrocytic (presumptive exo-erythrocytic) forms are inhibited, but eradication does not regularly occur. The only other drug which has a similar action is plasmoquin, but this drug has to be given in a dosage which is too dangerous for routine use in man".[5]

Suppression Therapy

Like mepacrine, paludrine is an efficient suppressive. It is quite probable that paludrine will prove far superior to other drugs in suppressive therapy by permitting such therapy to be given only once weekly. This idea derives its support from the remarkable effect of a single dose of the drug in controlling clinical attacks of *P. vivax* infections and in preventing subsequent relapses indefinitely if only weekly doses of 100 mg. of paludrine are sustained.[4,5] This matter will be considered later in detail.

Treatment of Clinical Attacks

Paludrine produces clinical cure of acute attacks of all types of malaria, as gauged by its action on the paroxysms and temperature and by the disappearance of asexual parasites from the peripheral blood. This is true both for induced and naturally occurring malaria. The drug was given by mouth.

In *P. vivax* infections paludrine proved effective over a wide range of doses, 10 mg. to 750 mg. twice daily for 14 to 28 days. There was rarely more than one rise of temperature above normal subsequent to the beginning of treatment. In most cases the asexual parasites had disappeared from the peripheral blood by the fourth day of treatment, and the sexual parasites by the fifth day. The effect of paludrine on the *P. vivax* parasites and the clinical cure of the disease was similar to that of adequate mepacrine dosage.

The therapeutic activity of the drug in *P. falciparum* malaria was investigated in doses ranging from 50 mg. to 600 mg. twice daily for 14 days. The clinical recovery was

usually as rapid as in benign tertian malaria, and the action on the asexual parasites was as efficient as in the case of *P. vivax*. The sexual parasites, when present, remained in the peripheral blood throughout the treatment.

These investigations of the Liverpool workers[4] have been confirmed in Australia.[5] Clinical cure was obtained with adequate dosage in acute attacks of *P. vivax*, *P. falciparum* and *P. malariae* malaria in both induced and naturally acquired infections. Besides, in induced *P. falciparum* malaria radical cure was obtained. The Australian workers also found that clinical cure was brought about in a group of induced (trophozoites- and sporozoite-induced) *P. vivax* infections after a single dose of 100 mg. of paludrine. The Liverpool workers[4] have now demonstrated, in agreement with this observation,[5] that acute attacks of benign tertian malaria can be terminated by the administration of a single dose of paludrine (50, 100, 200, 300 or 400 mg.). The effect of any one of these doses on the clinical course of malaria is rapid and almost as fast as it is with multiple twice-daily dosage. The patient feels better within a few hours of taking the drug, and, although, there is usually a rise of temperature above normal on the "paroxysm day" immediately subsequent to the treatment, this fever is not usually associated with a rigor. There is usually no further rise of temperature until relapse occurs in due course. The asexual parasites disappear more slowly from the peripheral blood than they do after repeated dosage, but the blood is usually clear of parasites by the fourth or the fifth day. Sexual parasites disappear more slowly than the asexual parasites.

The Problem of Relapse

It has already been noted that in induced *P. falciparum* infections radical cure was obtained with paludrine.[5] Paludrine is apparently more successful in eradicating *P. falciparum* infections. Evidence at present available at the hands of the Liverpool workers[4] seems to suggest that after dosage of 100 mg. twice daily for 14 days or more, subsequent relapses do not occur. In the dosage investigated (50 or 500 mg. twice daily for 14 days) paludrine showed no greater effect on the relapse rate of *P. vivax* malaria than full doses of mepacrine and is therefore less efficient than a combination course of quinine and pamaquin. With the single dose technique employed in benign tertian malaria parasites reappear in the blood 3 to 8 weeks after the administration of the dose, usually accompanied by clinical signs of malaria. However, such relapses respond immediately to a further single dose of the drug, a dose of 100 mg. being sufficient to abort the attack, independently of the size of the previous dose. It has been further found by the Liverpool workers that administration of one dose of 100 mg. weekly after treatment of the acute attack with a single dose of 50 to 400 mg. keeps the patient free from relapse indefinitely. Similar observations have also been made by the Australian workers.[5] However, in the actual treatment of an acute attack of relapse there is little to choose between single doses of paludrine and mepacrine, apart from the slight abdominal discomfort produced by the latter.

It has been remarked earlier that the secondary exo-erythrocytic forms, demonstrated in some species of bird malaria, is presumed to be present in *P. vivax infections* in man and to be the cause for relapse. Paludrine, but not mepacrine or quinine, has an action on these exo-erythrocytic forms, an action shared to some extent by pamaquin which is also known to lower the relapse rate in benign tertian malaria. Arguing by analogy and the evidence available at present[3] it is suggested that under suitable conditions paludrine may act on the as yet unidentified exo-erythrocytic forms of human *P. vivax* parasites. And it is hoped that the process of exposing the parasites to a long series of intermittent dosages of paludrine, as is done in the treatment of relapses, may prove more effective in producing radical cure of *P. vivax* infections than the continuous fortnightly twice-daily dosage regimes originally adopted.[4]

Action on Gametocytes

Paludrine has no direct effect on the production of gametocytes of either *P. vivax* or *P. falciparum*.[4,5] It has been found, however, that the gametocytes failed to develop in the mosquito if the patient's blood was ingested by the insect while he was taking the drug. A week after the cessation of the drug (by which time most of it would have been excreted from the body) the gametocytes in the patient's blood grew to maturity when ingested by the mosquito. The Australian workers[5] observe that paludrine acts on the developing gametocytes after it has been ingested by the mosquito and not while it is in the human blood stream. Although it is not truly gametocidal paludrine thus prevents mosquito infection during a therapeutic course and subsequently, until the blood level falls below the effective concentration. It is superior in this respect to both quinine and mepacrine.

The Effective Blood Concentration

The concentrations of paludrine in blood will depend on the rate of absorption and excretion of the drug. Paludrine is quickly absorbed from the gastro-intestinal tract, as would appear from the rapid effects it produces on acute attacks of malaria after oral administration—after a single dose of the drug the patient feels better within a few hours of taking the drug. The drug is also rapidly excreted in the urine. However, in spite of its rapid excretion, an effective blood concentration of the drug has been found to be maintained for more than 48 hours after a single dose of 200 mg. or more. The effective blood concentration is, at present, taken to be the concentration of the drug attained in the plasma after a single dose of 50 mg., the lowest dose so far studied and found to be effective in benign tertian malaria. Single-dose therapy may, therefore, be regarded as a method of providing effective drug concentrations lasting not just for few hours but for several days.[4]

Toxic Reactions

Paludrine is effective, as has been already noted, over a wide therapeutic range, and the difference between the effective therapeutic range and the toxic dose is again considerable. Although abdominal discomfort and nausea, and sometimes vomiting half an hour to an hour after taking the tablets were noticed in some patients receiving 500 mg. or more twice daily; in no case, however, was it necessary to stop the treatment because of toxic symptoms. No urinary symptoms were observed on any dosage schedule. Doses upto 750 mg. twice daily did not give rise to any serious side effects provided the patient was given fluid while taking the drug. Patients lying quietly in bed were found to be more tolerant to the drug than volunteers taking the drug while actively engaged in their ordinary work.[4] After doses of 1000 mg. a day only minor side effects were noted by the Australian workers.[5]

Summary and Concluding Remarks

(*i*) Sporozoites are apparently unaffected by paludrine.
(*ii*) Paludrine exerts a complete lethal action on the

primary tissue phase of falciparum infections and a partial effect on vivax infections.

Paludrine is a perfect causal prophylactic in falciparum infections and is an efficient suppressive in either.

(*iii*) Paludrine is schizonticidal, producing remarkable effects in controlling acute attacks of all types of malaria and radical cure in falciparun infections.

(*iv*) The secondary tissue phase which is presumed to exist in man and thought to be the cause for relapse in benign tertian malaria, is probably affected in man and it is hoped that by proper technique of dosage and procedure radical cure may be ultimately possible, or the relapse rate considerably reduced.

(*v*) The gametocytes, although unaffected in the patient's blood, fail to develop in the mosquito when they are ingested by the insect while the carrier is on a therapeutic regime.

The discovery of paludrine has been remarkable in many respects. The drug represents an important advance in the field of synthetic antimalarials, developed, as it is, along a hitherto unknown chemical line. No other known antimalarial drug combines so many desirable properties in one. The drug constitutes the first practical causal prophylactic and one of its pre-eminent therapeutic achievements is that a single dose of the drug will terminate an acute attack of malaria, and if continued in weekly doses will keep relapse of benign tertian malaria indefinitely postponed which, it is hoped, may finally lead to radical cure. This achievement of the drug lends itself easily to be used as a suppressive, and suppressive therapy in itself may, if properly dosed, render the blood of the carrier of sexual parasites non-infective to mosquito, an important fatcor in malaria control. The drug is colourless, virtually non-toxic and is easily taken by mouth—properties which make it admirably suited to prolonged mass therapy. The potentialities of paludrine in malarial chemotherapy are yet to be fully revealed, and with wider use its undesirable effects and disadvantages may be exposed as much as the therapeutic range is expected to be extended. As for instance, a combination course of paludrine and pamaquin both of which show an action on the secondary tissue phase may provide the answer to the vexed problem of relapse in benign tertian malaria. Unfortunately, however, the drug is not available at present for general use. It may be noted here that experimental and clinical investigations are being conducted under the guidance of Prof. B. N. Ghosh who has received a supply of paludrine for the purpose from Dr. D. G. Davey, a very prominent member of the team that discovered paludrine.

Acknowledgments

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CURRENT MEDICAL LITERATURE

Colloidal Aluminum Hydroxide in the Treatment of Peptic Ulcers

Collins (*J. A. M. A.*, **127**:899, 1945) from the analysis of the results obtained by the administration of colloidal aluminum hydroxide in more than 3000 cases of peptic ulcer treated in the Cleveland hospital during the past 8 years writes that each patient should be considered separately and treated according to the condition and his alimentary habits. The patient is placed in the first week on a bland diet but meat, cooked vegetables and fruit are added during the second week and within a month he has a well-balanced diet except for the avoidance of seeds, skins and coarse fibres. Colloidal aluminum hydroxide is administered every 2 hours during waking hours in the first month's treatment and on alternate hours with the feeds so that every hour the patient takes either food or colloidal aluminum hydroxide. The size of the dose depends on the degree of the gastric acidity and the severity of the symptoms but is never less than 2 drachm (8 c.c.) and the bedtime dose is never less than 1 ounce (30 c.c.). In the case the patient has severe night pain the bed-time dose is repeated at midnight and at 3 a.m. during the first week's treatment.

For a simple ulcer, not complicated, the author advises the use of colloidal aluminum hydroxide 6 times a day from the beginning of the treatment. The method is to be applied during the first two months; for the rest of the year, the method of alternate alimentation is to be used with colloidal aluminum hydroxide twice a day, once after the day meal and once at bed-time.

Apart from the acid combining power and the power of inactivating pepsin, the alumina gel has advantage over the usual alkalis. Its astringent and demulcent properties are of value and unlike sodium bicarbonate, it does not cause a compensatory stimulation of acid secretion. It is not absorbed and can, therefore, be given without the hazards of alkalosis. It does not constipate. No trouble of acid-base equilibrium or urinary complications have been observed in the cases so treated.

The periodic controls through radioscopic examination and even by gastroscopy has shown most satisfactory results in way of progressive disappearance of the ulcer.

Cardivascular System in Anaemia

Wintrobe (*J. Hæmat.*, **1**:121, 1946) reviews the cardiovascular and physiologic adjustments which occur in the presence of anæmia. These include increase in the cardiac rate, velocity of blood flow, minute volume, cardiac output, cardiac size, oxygen utilization and oxygen consumption; and decrease in the circulation time, the blood viscosity, the arterial blood pressure, the total blood volume, the A. V. oxygen difference, and the vital capacity of the lungs. He suggests that the remarkable changes found in the cardiovascular system in cases of sickle cell anæmia may be the result of adjustment to severe anæmia of exceptional chronicity.

Dehydration Pleuritis

The phenomenon of tissue dehydration continues to arouse interest as a causal or contributing factor in many clinical syndromes. Armanino and Ory (*Am. J. M. Sc.*, **211**:597, 1946) have shown that pleural injury with fibrinous exudate as judged by respiratory pain and friction rub may occur as

one of the complications of dehydration in diabetic precoma. While cultures of the pleural exudate were not made, the absence of fever and the prompt disappearance of pain and friction rub on rehydration indicate that the authors were dealing with a so-called sterile pleuritis in which the relative lack of interpleural and intrapleural liquid played an important part. Doubtless a review of old necropsy records of the pre-insulin era may disclose negative cultural results of fibrinous serous exudates in the severely dehydrated patient. Infection, of course, is not a *sine qua non* of the inflammatory reaction. Cell necrosis is all that is needed; dehydration should be sufficient in some cases to produce death of pleural mesothelial cells. The process of reaction and repair includes the laying down of fibrin. With water and electrolyte balance restored, the visceral and parietal pleural surfaces would be normally separated and restoration of the intact serous membrane quickly achieved. The abdominal pain so frequent in diabetic acidosis may reflect a peritoneal injury based on a similar hypothesis. An "aseptic" pericarditis has frequently been noted in fatal cases of chronic nephritis. The broad classification of chronic nephritis into two main types, nephritis with œdema and nephritis without œdema (Christian), suggests that the nephritis with dehydration due to increased permeability of the kidneys to water while witholding toxic nitrogenous products, offers conditions for cell injury in loci where movement of surfaces requires lubricating liquid. The pericarditis in chronic nephritis might be so explained. Another possible site exists in joint membranes where the synovial fluid may be so depleted in dehydrating conditions that the synovial cells are injured; if weight is applied, a "dry" arthritis ensues. Some arthropathies, in the light of the work of Armanino and Ory, need careful appraisal of the role that dehydration may play in their pathogenesis.—*J. A. M. A.*, **131**:1214, 1946.

Pulmonary Amoebiasis

Chaudhuri and Rai Chaudhuri (*Indian M. Gaz.* **81**:67, 1946) write that amœbiasis of the lung is much more frequent than is usually thought, but it often remains undetected, or perhaps is wrongly diagmosed as pulmonary tuberculosis. Clinical recognition of the condition is rarely reported, while the radiological findings, if present, are apt to be missed or misinterpreted.

Both the cases, reported by the author are of considerable interest and practical bearing. There was no history of dysentery in the past; there was no hepatic enlargement and no E. *histolytica* was found in the faeces. The fever, cough, pain in the chest, high leucocytosis, a mottled opacity in the lung field and the striking response to the therapeutic test with emetine were the main factors on which the diagnosis of pulmonary amœbiasis was arrived at in the first case. In the second case fever and hæmoptysis were the presenting features; E. *histolytica* were found in the sputum, and there was good response to specific treatment. In both the cases there was aggravation of symptoms after the first few injections of emetine but the ultimate results were remarkable. It is well to remember this condition where an obscure fever with lung symptoms cannot be explained.

Amoebic Pericarditis

Kern (*Med. J. Australia*, **1**:159, 1946) writes that the involvement of the pericardium is rare in amoebiasis, and this alone is sufficient reason to draw attention to the case reported below.

The patient was a seventeen-year-old Negro. For three months prior to admission to hospital he had suffered from intermittent pain in the right shoulder, malaise and progressive loss of appetite and weight. For two months he had had pain in the lumbar region, being particularly severe in the left. For three weeks he had had fever and profuse night sweats, and occasionally pain in both side of the chest, but no cough. For a week there had been pain in the umbilical region and sharp pain in the left flank radiating towards the left axilla. There was no history of diarrhoea. The patient was gravely ill. The temperature and pulse rate were elevated and the blood pressure was low. Examination showed that the heart was slightly enlarged to the left, with a loud pleuro-pericardinal friction rub and a thrill over the lower left side of the precordium. A few rales were heard over the bases of both lungs, especially the right. There was tenderness in both flanks and costovertebral angles, especially the left. The liver was palpated four fingers' breadth below the costal margin, but was not tender. Blood examination revealed a neutrophile leucocytosis and mild anæmia, the blood sedimentation rate was considerably raised, and a tuberculin test gave a positive reaction. On the sixth day after the patient's admission to hospital he complained of sharp precordial pain. Next day this became severe and circulatory collapse developed. X-ray examination now showed considerable increase in the size of the heart shadow and a small area of increased density in the right costophrenic angle. Six hundred cubic centimetres of thin chocolate-coloured fluid were withdrawn from the pericardimum and dramatic improvement in the patient's condition occurred. The fluid contained many red and white blood cells, the majority of the latter being neutrophile cells. Similar large amounts of fluid were withdrawn from the pericardium on a number of subsequent occasions. The fluid became progressively thicker and its cell content less. Its colour changed from chocolate to brick red. Repeated search for bacteria, including the tubercle bacillus, by smear and cultural examination was fruitless. No parasites were seen in several wet preparations examined. On the ninth day in hospital the right pleural cavity was found to be filled with clear straw-coloured sterile fluid, which rapidly reaccumulated after removal. On the twenty-seventh day in hospital the patient had a gross haemoptysis. He died ten days later.

The opinion held was that the cause of death was tuberculous pericarditis. Autopsy, however, revealed that the underlying infection was amoebiasis. The pericardial surfaces were yellowish-grey and granular, covered by a thick layer of nectrotic material backed by granulation tissue. Similar shaggy, friable, necrotic material covered the surfaces of the right pleural cavity. The right lung contained nuemrous abscesses, many connecting with bronchi. In the right lobe of the liver, immediately below the diaphragm, was a large abscess filled with friable, yellowish-green material and with a firm greyish wall. No connection could be demonstrated between the hepatic abscess, the pleural cavity or the pericardium. The necrotic tissue in the liver abscess on the pericardium and pleura and in the lung abscesses and the walls of the liver abscess contained a great number of amoebae, many containing red blood cells.

Difficulty in Diagnosis in Pericarditis

Hanneson (Tubercle, **22**:79, 1941) opined that serious disease is nowhere more liable to be overlooked than when

it affects the pericardium. Certainly tuberculous pericarditis, with its insidious onset and its confusing early symptoms, may easily escape diagnosis, and is probably more common than is generally believed (White—Heart Disease, New York, 1944 and Ellman—*Brit. Heart* J., **7**:147, 1945). The onset, unlike that of rheumatic pericarditis, is seldom associated with pain; the accumulating of fluid, being gradual, is well supported, and a very large effusion may form before attention is attracted by signs of mediastinal pressure: indeed as much as 2-3 litres has been aspirated. The fluid is more often blood-stained than serous, and it may be difficult to demonstrate tubercle bacilli either by culture or by guineaping inoculation. A further cause of difficulty is that the constitutional disturbances of active tuberculosis which attend the pericarditis itself may easily be attributed to the primary lesion in the lung, pleura or spine, and diagnosis is then delayed until the development of cardiac tamponade. Sometimes, however, the condition arises in a patient (of any age) who has been in good health until a recent influenza-like illness; in these cases infection of the pericardium is attributed to direct lymphatic spread from reactivated tuberculous mediastinal or hilar glands; and prompt diagnosis is particularly important since the primary treatment is that of the underlying tuberculous focus.

If the patient survives, he develops the features characteristic of Pick's disease—enlarged liver, ascites, muffled heart-sounds, and small pulse-pressure—for which surgical treatment now offers the chance of dramatic cure. It may be that in future early recognition of tuberculous pericarditis and improved treatment will arrest the disease before this stage of constrictive percarditis is reached. The induction of a pneumopericardium, already helpful in diagnsos, may also find a place in therapy. The value of local penicillin in acute cocal pericarditis is already established (Wise and Shefter—*J. A. M. A.*, **127**:583, 1945). Is it too much to hope that antibiotics of the streptomycin group will provide the remedy for a condition which has hitherto resisted active medical treatment? —(Lancet, **2**:245, 1946).

The 'Burning-Feet' Syndrome

Gopalan (*Indian M. Gaz.*, **81**:22, 1946) writes that the patients showed obvious evidence of malnutrition. Signs suggestive of riboflavin deficiency were afmost invariably present.

A burning sensation in the extremities is the characteristic feature of these cases. It is usually confined to the soles of the feet, but is present in some cases, in the palms of the hands as well. Characteristically, the sensation of burning starts in the soles of the feet round about the ball of the great toe and seems to spread therefrom to involve the soles completely. In some cases, the burning sensation may spread upwards to involve the dorsum of the feet and the ankle region.

The patient also complains of a feeling of 'pins and needles' in the distal parts of the extremities. This is distinct from the burning sensation referred to above. It is mostly felt on the dorsum of the feet and round about the ankles.

In striking contrast to the marked subjective symptoms is the paucity of objective signs, even in cases which have had the disease for over 2 years. The limbs do not show any wasting. Foot-drop, so commonly seen in advanced and neglected cases of peripheral neuritis, is never observed in these cases. The limbs are not spastic and their muscular power is not diminished. The knee jerks are brisk, the abdominal reflexes are preserved and the plantar response is flexor. There is no loss of sensation of any kind; touch, temperature and pain sensations, the vibration sense, the sense of position and passive movement are all retained even in advanced cases. A striking feature is the absence of tenderness in the calf muscles.

An interesting feature observed in several cases is hyperidrosis of the affected parts.

Thirteen cases were treated with 4 to 6 drachms (15 to 22½ grams) of vegemite twice daily for periods ranging from 3 to 4 weeks. Definite improvement was obtained in all the 13 cases; the burning sensation and the feeling of 'pins and needles' subsides, and by the end of the fourth week of treatment the subjective symptoms had completely disappeared in all cases.

Thiamine in a 50 mg. dose daily by injection was tried on a group of 10 cases for a period of about three weeks. No improvement was obtained in any of these cases. Nicotinic acid (300 mg. daily by the oral route in 4 cases and 100 mg. daily by the parenteral route in 4 other cases), given over a period of about three weeks, was equally ineffective in inducing improvement. A group of 12 cases was treated with 10 mg. of riboflavin daily by mouth for periods ranging from 2 to 3 weeks. With this treatment there was rapid subsidence of the associated signs—glossitis, angular stomatitis, ocular symptoms, etc., but the burning sensation continued with unabated severity; indeed a few patients reported that this was worse at the end of treatment.

The response to treatment with pantothenic acid was investigated. The cases were treated for periods ranging from 2 to 3 weeks with 20 to 40 mg. of calcium pantothenate injected intramuscularly daily. Definite improvement was obtained in all the ten cases. The improvement was more striking and rapid than that obtained with vegemite; indeed in cases where 40 mg. of the drug daily were employed, the patient reported improvement within the 5th day of treatment, and the improvement was complete by the end of a fortnight of treatment. The patients tolerated injections of calcium pantothenate well; no untoward reactions were noticed when even 80 mg. of calcium pantothenate were administered in a single dose by injection.

Conclusions: A group of 53 cases of 'burning feet' has been investigated. The condition was found in close association with signs of ariboflavinosis.

The symptoms were relieved by marmite therapy, and by injections of calcium pantothenate, but were not influenced by treatment with thiaminnictotinic acid and riboflavin.

The differences between the syndrome and peripheral neuritis are discussed, and the probable nature and site of the lesion in cases of 'burning feet' is also discussed.

Some not well-known Manifestations of Riboflavin Deficiency

Gopalan (*Indian M. Gaz.*, **18**:227) writes that the occurrence of genital symptoms in women suffering from riboflavin deficiency is not so rare as the scantiness of the reports on them indicate.

Leucorrhoea was present in several women suffering from orolingual and other signs of riboflavin deficiency observed in the clinic. Treatment with riboflavin in these cases resulted in improvement of the orolingual condition and simultaneous subsidence of the vaginal discharge. The significance of

leucorrhoea in women is hard to assess, considering the variety of conditions that may produce it. Careful examination to exclude other possible causes is necessary before attributing the disorder to riboflavin deficiency. Where other causes are ruled out by careful examination, the occurrence of leucorrhoea in cases suffering from accepted signs of riboflavin deficiency and the subsidence of the leucorrhoea on the administration of riboflavin are significant. It may be justifiable to infer in these cases that deficiency of riboflavin was the cause of the leucorrhoea.

The present investigation showed that genital symptoms in females, suffering from riboflavin deficiency, occur predominantly in women of the child-bearing age; they were rare in infancy, childhood and old age. Out of 67 cases which presented genital symptoms in association with other signs of riboflavin deficiency and in which these symptoms cleared on the administration of riboflavin, 64 were adult women and only 3 were children.

The minimal genital manifestation of riboflavin deficiency in women is an increase in the normal vaginal secretion. Low-grade secondary infection may often be superposed on the devitalized vaginal mucosa and contribute to the aggravation of the leucorrhoea. Alternatively a primarily non-nutritional leucorrhoea may be made worse by a coincident riboflavin deficiency.

Among adult women suffering from riboflavin deficiency, *pruritus vulvae* is present in cases with profuse leucorrhœa.

In children, genital signs are often absent even in cases of severe and advanced riboflavin deficiency. Out of 89 children with signs of riboflavin deficiency observed in the course of the investigation, only 3 children showed genital signs. All these three suffered from vulvitis, which in one case was so severe as to involve the labia and surrounding skin. Out of 131 adult women with signs of riboflavin deficiency observed in the same period, 64 exhibited genital signs; in 39 of these leucorrhœa with varying degrees of congestion of the vaginal mucous membrane was present, and, in the remaining, in addition to the above, pruritus vulvae with varying degrees of vulval inflammation was encountered.

Fissure anus, pruritus ani and perianal inflammation have been mentioned among the signs of riboflavin deficiency. Several patients suffering from signs of ariboflavinosis complain of burning pain during and after defaecation. Examination of these cases reveals the presence of one or more fissures at the anal margin. Patients suffering from signs of riboflavin deficiency are, not infrequently, constipated. The passage of hard scybalous faeces aggravates the fissures.

It is possible, however, that in several cases of fissure anus associated with evidences of riboflavin deficiency, the nutritional defect may not be the sole cause of the fissure; the underlying riboflavin deficiency might have simply aggravated the anal condition and prevented the fissures from healing with the usual methods of treatment.

Treatment of all these cases with 10 mg. riboflavin daily by mouth for 2 to 4 weeks leads to rapid healing of the fissures and subsidence of other signs.

Prepucial ulceration—The condition of scaly dermatitis of the scrotum which is frequent in subjects of riboflavin deficiency is well known. The free margin of the prepuce in several men showing signs of riboflavin deficiency appears white, sodden and ulcerated. Pruritus is often associated with this condition. The condition clears up rapidly on the administration of riboflavin.

An interesting observation is that these genital manifestations are strikingly rare in children. They occur mostly—almost always—in adults.

Angular blepharitis—In several cases of riboflavin deficiency observed here, the angles of the lids showed ulceration which involved both the cutaneous and the conjunctival aspects of the lids. The skin of the lids around the affected region appeared white and sodden. Both angles of the lids were often involved but sometimes the medial angles alone showed the lesion. This condition which is predominantly an affection of the lids and hence perhaps more aptly termed 'angular blepharitis' clears up rapidly on treatment with riboflavin.

In conclusion, it may be emphasized that the clinical manifestations of riboflavin deficiency are to be found not only in the eyes and mouth but in other mucous and mucocutaneous parts of the body—in the vulva and vagina, the prepuce and the anal and perianal regions.

Low-Sodium Diet and Free Fluid Intake in the Treatment of Congestive Heart Failure

Bridges and others (*New England J. Med.*, **234**:571, 1946) write that the treatment of cardiac œdema by restricting sodium and permitting a free fluid intake has not received wide application, although its usefulness is now generally appreciated. At the Massachusetts General Hospital, sixty-four patients who had congestive heart failure were treated by this technique as well as with digitalis, diuretics, and other routine measures. Seventeen patients obtained much help, fifteen moderate benefit, eight slight benefit, and seven no benefit. In the remaining seventeen cases, the patients were either unco-operative or the data were insufficient to warrant inclusion in the present report.

The diet used contained about 700 mg. of sodium in an amount of food equivalent to 1,800 calories. If the food tasted too flat, ammonium chloride was used in place of salt. It was essential that no salt or soda be used in cooking or included in incidental medicines.

The advantages of the treatment are that it frequently permits control of œdema that cannot be controlled otherwise, diminishes the frequency with which mercurial diuretics must be given, and enables the patients to take more water. The disadvantages are the difficulty of preparing or obtaining the diet outside of a hospital and the taste of the food.

The optimum amount of water to be taken has not yet been determined and it is uncertain whether 5,00 to 6,000 c.c. per day is more beneficial than 2,000 to 3,000 c.c. Undoubtedly, however, the water intake formerly recommended was distinctly inadequate because diminished renal function often prevents sufficient excretion of waste products when the daily fluid intake is less than 1500 c.c.

Fluids in Congestive Heart Failure

Levy and others (*J. A. M. A.*, **131**:1120, 1946) write that a study of 122 patients with congestive heart failure was conducted specifically to evaluate the relative merits of restricting fluids, allowing fluids *ad libitum* and forcing fluids; they observe:

Of 36 patients observed on restricted fifluids, 27·7 per cent complained of thirst (52·6 per cent of those observed during the summer); 13·6 per cent of patients discontinued restriction because of thirst. Restricted fluids may lead to dehydration with disorientation.

On fluids *ad libitum* the average cardiac patient consumed approximately 1,700 c.c. daily during the summer, 1,300 c.c. daily during the winter and 2,200 c.c. daily with febrility, without ill effects in any instance. Patients on fluids *ad libitum* were much more comfortable than members of the other groups studied.

Of 38 patients observed on forced fluflids, 18·3 per cent were nauseated, necessitating discontinuance. Of 31 adhering to the regiment the results were good.

In general, compensability is independent of fluid therapy. No effect of fluid therapy on mortality was noted.

Digitoxin or a similar pure glucoside is the preparation of choice for routine digitalization. The acid ash salt poor diet is the diet of choice in treating congestive heart failure.

Essential therapy in congestive heart failure is the judicious employment of bed rest, digitalis, diuretics and sedatives, with an acid ash salt poor diet providing sufficient calories, minerals and vitamins.

With restricted sodium intake, restriction of water is unnecessary in treating cardiac decompensation, increases the disccomfort of the patient and may prove deleterious.

In most decompensated cardiac patients forcing fluids will neither retard nor facilitate compensation. When congestive failure is complicated by sepsis requiring chemotherapy, azotæmia or dehydration in associated medical or surgical conditions, fluids should be forced to obtain optimum therapeutic results.

As in health, the average patient with congestive heart failure should be allowed to drink water as it is desired, consuming enough to maintain a daily minimum urinary output. Water intake should be increased to prevent dehydration where there is intrinsically impaired renal function, sepsis, excessive skin or urinary water loss.

Curative Treatment of Lathyrism

Jacoby (*Indian M. Gaz.*, **81**:246, 1946) writes that lathyrism is a nervous disease of the upper motor neurone in its clinical aspect identical with spastic spinal sclerosis. But whereas for the latter disease no cause and no curative treatment is known, the cause of lathyrism is consumption of the seed *Lathyrus sativus* (Teora). With regard to treatment, it has been found that prostigmine, the synthetic equivalent of physostigmine, has definite curative properties if given as injection (1 c.cm. of prostigminemethyl sulphate contain, 0·5 mgm.) for a sufficient length of time. In this way were treated 14 patients altogether, eight of whom, *i.e.*, 57·1 per cent of the milder ones, were practically cured. Three, *i.e.*, 21·4 per cent, were greatly improved and three, *i.e.* 21·4 per cent of the far advanced cases, were not improved so far.

Peptic Ulcer Treated with Gastric and Intestinal Extracts

Hulbacher (*Lancet*, **2**:282, 1946) reports on the results of the treatment of 54 patients suffering from gastric or duodenal ulcers with gastroduodenal extracts. The idea behind this treatment is that normal gastric and duodenal function is maintained through the stimulus of various biologically active substances (such as enterogastrone) which are present in healthy mucosa and whose absence favours ulcer. The extracts used by the author are in his opinion specific in the sense that he was successful only when stomach extract was used for gastric ulcer, and small-intestine extract for duodenal ulcer, and not if the extracts were reversed. Of his 54 patients, mainly ambulant, most were given treatment by injection as well as by mouth, and did not leave off work, abide by any strict diet; and of these 54, all but 10 showed 'good results' over sixteen months.

All the cases which were benefited by this treatment showed rapid disappearance of symptoms, increase of weight, and rapid decrease in size of the ulcer niche as disclosed by radiography. Some cases of indurated ulcer reacted favourably, but others, and cases invovling tumours, were not influenced. Injection of the preparations gave rise to no undesirable secondary effects apart from occasional pain at the injection site. The administration of the extracts as tablets to supplement the injections and as prophylaxis is suggested.

Penicillin in Dementia Paralytica

Reynolds and others (*J.A.M.A.*, **131**:1255, 1946) write that forty-one patients with dementia paralytica have been treated with commercial penicillin: 24 with penicillin alone; 17 with penicillin and malarial therapy.

Penicillin alone, in doses ranging from 2·0 to 10·0 million units, produced at least some degree of clinical improvement in 11 out of 24 patients (46 per cent). Improvement in the spinal fluid abnormalities generally was apparent. As a rule the spinal fluid cell count and total proteins promptly became normal, and the colloidal gold and Wassermann tests gradually improved.

Penicillin administered concurrently with malarial therapy resulted in clinical improvement in at least 10 out of 17 patients (58 per cent). Improvement in the spinal fluid abnormalities was even more complete than with penicillin alone.

The effectiveness of concurrent penicillin-malaria therapy is such as to make it, for the present at least, the treatment of choice for patients with dementia paralytica.

Radio-Active Iodine in Toxic Goitre

Iodine was one of the first elements to be made radioactive artificially (in 1934), and for some years experiments have been in progress in the U.S.A. to determine its effects on the thyroid. It is known now that in a patient not under the influence of iodine 80 per cent of a dose of radio-active iodine is taken up in the thyroid within a short time of ingestion. It there gives off rays which generate high-speed electrons, and these affect neighbouring tissues to a distance of a few millimetres. This effect is the same as that ultimately produced by X-rays, but the internal radiation has two advantages in that it does not have to penetrate the skin and other overlying tissues, and that it is probably distributed evenly throughout the gland. Theoretically therefore, radio-active iodine should produce better results in toxic goitre than x-ray therapy, which in the opinion of most clinicians has not been a success, and this is verified by recent clinical reports. (Hertz and Robert,—*J. A. M. A.*, **131**:181, 1946) and Champan and Wan (*Ibid*, p. 86). The radioactive iodine is taken by mouth within a few hours of its preparation, as an almost tasteless liquid. Occasionally there are slight toxic effects, including pyrexia and nusea for a day or two. The patent must have taken no iodine for several weeks previously.

Of the 51 cases reported in the two papers it is claimed that four-fifths were restored to normal, the basal metabolic rate falling to normal in one to five months, and the gland shrinking in many cases to normal size. Fibrosis is produced in the gland by this method, as shown by biopsy. In 9 cases

hypothyroidism or myxoedema resulted, through in 5 this occurred only after a subsequent thyroidectomy, and in 2 of the others the B.M.R. rose spontaneously to low normal levels. The dose of radio active iodine is calculated in millicuries, and the strength required is proportional to the size of the goitre. Most of the patients required only a single dose, but others required two or three. The method has the advantage, from the patient's point of view, of extreme simplicity, and apart from the possibility of myxoedema it seems to be free from risk.—Lancet, **29**:166, 1946.

Para-Aminobenzoic Acid in Endemic Typhus

Smith (*J. A. M. A.*, **131**:1114, 1946) writes that a series of 29 patients with endemic (murine) typhus were given 2 gm. of para-aminobenzoic acid every two hours until their temperatures returned to normal. The number of days of fever in patients receiving this treatment was, on the average, significantly less than in a comparable series of patients not receiving the drug.

No toxic effects that could be attributed to para-aminobenzoic acid were observed in a group of 46 patients, each of whom received an average of 95 gm. of the drug. Twenty-nine of these patients had murine typhus.

It is recommended that a thorough clinical trial be made of this drug, using a larger number of patients under more rigidly controlled conditions. Such trials should include appropriate studies designed to detect an early toxic effects.

Prothrombin Time in Normal Indians

Braganca and Rao (*Indian M. Gaz.*, **81**:245, 1946) write that the prothrombin time in normal Indians, both men and women, has been established by the macro and micro methods. No difference is found in the prothrombin time of men as compared with that of women.

The normal range by the macro method is 15 to 29 seconds, while the range by micro method is 18 to 25 seconds.

A comparison of the prothrombin time of patients having positive Wassermann and Kahn tests with normal controls, shows that there is no difference in the two groups even when the dilution method is applied.

The Blood Pictuure of Radiologists

Mendonca (*J. A. M. A.*, **131**:1240, 1941), hematologist of the army medical corps, in a paper recently published covers the results of his personal study of the blood picture of twenty-one radiologists of Reo De Janeiro. The study disclosed 16 cases of anæmia, normal conditions being found in five radiologists. Out of these 16 cases of anæmia, 11 cases were of the hypocytaemic, normochromic and normocytic type, 4 cases were of the hypocytæmic, normochromic and macrocytic type and 1 case was of the hypocytæmic, hyperchromic and macrocytic type. The anæmia was always of slight intensity, with only 1 case of the pernicious form, and no case of the hypochromic, microcytic type. It was not possible to confirm the x-ray erythrocytosis described by Lavedan, Portis, Pfahler and Mottram, the cases studied being closer to the types characterised by Beclere and Moldawsky. It was not possible either to establish any definite association between the intensity of the anæmia and the total time of work (varying from one to twenty-five years), as well as with the special kind of work (fluoroscopy, radiography or roentgen therapy), the type of equipment and the type of protection. Of the 21 persons, only 7 are subjected to periodic blood examination, and 8 do not take regular vacation periods away from the x-ray work. In only 3 cases of anæmia was the number of reticulocytes abnormally high, the rest, with or without anæmia showing a normal reticulocyte count. This is contrary to the opinions of Moldawsky and of Gerard, who emphasized that reticulocytosis is a characteristic of radiologists' blood. In reference to the white cells the findings were normoleukocytæmia with normoneutrocytæmia and moderate nuclear deviation (Schilling index 1/6), slight relative eosinocytæmia and slight relative lymphopenia and normomonocytæmia. The author points out, as one important conclusion of his study, the absence of the leukæmia producing effect of roentgen radiation in spite of the presence of some important contributory factors, as, for instance, long and active work with x-rays which was recorded in some of the cases. The author believes that this may be explained by a lesser sensitivity of the hæmopoietic organs of these persons, similar to the well known fact of the low incidence of agranulocytosis caused by the sulphonamides and other drugs in Brazil.

Ocular Manifestations of Avitaminosis in Adults

Sardana (*Indian M. Gaz.*, **81**:188, 1946) from an analysis of 500 cases of avitaminosis with ocular symptoms groups them under the following heads:—

Vitamin A deficiency—Out of the 500 cases forming the basis of this article, 159 suffered from vitamin A deficiency. The chief complaints of these cases were night blindness. Itching of the eyes, and a feeling of dryness were frequently admitted. About 3 per cent cases had one or more meibomian cysts in the lids and varying degrees of blepharitis were present in 5 per cent cases. The cornea was insensitive in 81 per cent of cases and keratinizing epithelial metaplasia was observed in some of them. A few cases of disciform keratitis (2 per cent) and few more with sclerosing keratitis (2 per cent) were seen. Only one case in the whole series had phlyctenular kerato-conjunctivitis. The resulting opacities in all these cases, as well as in those with corneal ulcers, became faint as the acute stage was over but did not disappear completely. About 2 per cent cases developed corneal ulcers. These responded well to local application of red palm oil. They manifested marked lacrymation and photophobia, but pain was remarkably absent altogether or was very slight indeed. Not a single case showed xerosis or keratomalacia. The pupils tended to be dilated in all these cases, but the reaction to light was brisk. Ocular tension was practically normal in all caess. The fields of vision (tested by hand movements) showed marked concentric contraction, with what amounted to 'tubular' vision in advanced cases. Visual acuity for distance was found reduced in all cases.

Night blindness in these cases preceded the failing of vision in day. In dark nights the patient could not see the ground in front of him clearly, and could not distinguish elevations and depressions; looking at the sky, however, he could see the stars more or less clearly; moonlight afforded him some relief in moving about; bright electric light gave the best vision, but adaptation to darkness was greatly delayed.

Reading and near vision in early stages was apparently unaffected; in later stages, however, the patients had some difficulty in reading, particularly in dim light.

Ophthalmic examination revealed that the media were quite clear and the fundi appeared normal.

Thiamin (vitamin B_1) deficiency—Two hundred and twenty out of the 500 cases under report were thiamin

deficiency cases. Their chief ocular complaint was *dimness of vision*—inability to recognize faces from a distance of 20 or 30 yards. Literate patients complained of difficulty in reading.

A burning sensation in the eyes, a dull pain in the back of the eyeball, discomfort on exposure to sunlight, lacrymation, mild photophobia with some degree of blepharospasm were present in almost all the cases.

The conjunctiva and cornea were practically normal in appearance; the pupils were normal in size or slightly contracted, briskly reacting to light but sluggish or inactive to accommodation, the reverse of the Argyll Robertson pupil. Intra-ocular tension tended to be increased.

Visual fields showed mild peripheral contraction; central visual acuity was reduced—the reduction was more marked than the contraction of the field of vision; accommodation power was weak, and near vision was consequently tiring, simulating premature and rapidly progressing presbyopia. Some patients were actually using rather indiscreetly prescribed convex glasses for reading and close work.

Ophthalmoscopic examination revealed clear media and normal fundi except in 2 per cent of cases manifesting signs of optic nerve atrophy.

Visual disturbance in all cases, both for distanct and for near work, was equally marked during day and night.

Riboflavin deficiency—Cases of riboflavin deficiency, 121 in the 500 under report, complained chiefly of hazy vision in the sun.

All the cases denied having any pain or itching in the eyes, but some admitted having a burning sensation. In practically all cases, the conjunctiva showed a streak of vascular injection in the interpalpebral region, extending from limbus literally to the outer canthus. At the outer canthus were also seen superficial cracks extending to the cutaneous surface. These gave an appearance of angular conjunctivitis.

The cornea was found clear in all cases except one, which showed sclerosing keratitis with circumcorneal injection. This injection and the corneal vascularization cleared under treatment, but localized 'haze' in the deep layers of cornea persisted.

The pupils were moderately dilated and sluggish in reaction to light unlike the pupils in vitamin A deficiency. The visual discomfort in the sun and bright light may be attributed to dilated sluggishly reacting pupils.

The central visual acuity for distance was reduced, but the accommodation power was practically normal, and reading and close work could be attended to without much difficulty.

Ophthalmoscopy revealed no abnormality either in the media or in the fundi. The fields of vision showed contraction in the upper and temporal regions but were normal in the nasal and lower regions in the early stages; in advanced cases the contraction of the fields of vision involved the lower and nasal regions as well, and ultimately the contraction was marked in all regions so that it resembled the contraction in vitamin A deficiency cases. At this stage the parties complained of impairment of vision in night as well.

Gastric Carcinoma: Review of Errors in Diagnosis

Meyer Golob (*Am. J. Digest. Dis.*, **13**:17, 1946, Ref. Radiology, **47**:535, 1946) lists four factors which enter largely into the wrong interpretation of symptoms and lead to delay in the diagnosis of gastric cancer: (1) age incidence; (2) length of history; (3) size or site of lesion; (4) malignant degeneration of a benign ulcer. Neither the age of the patient, the size of the lesion, nor the presence of hyperchlorhydria or achlorhydria should lessen the suspicion of gastric cancer. Nor should dependence be placed on a therapeutic test, since practically every case shows relief of symptoms when clinically treated.

To illustrate the fallacy of ruling out cancer because the patient is "too young", a case is recorded in a man of 37 with a history of gastric symptoms for eight years. He was first seen by the author in January 1936, with complaints dating back two years—loss of weight, anorxia, and hypochlorhydria progressing to achylia. Roentgen examination in December 1934 had shown an irregular antrum and bulb which were thought to be due to an old ulcer. Exploration was recommended, but the patient improved on a medical regime, and operation was postponed until January, 1936, when an anaplastic grade IV carcinoma was found, with metastases in the regional lymph modes.

The cases are presented to show that a short history does not necessarily rule out cancer. The first patient, a 64-year-old man, showed the taxtbook picture of malignant neoplasm, though earlier roentgen examination in a commercial laboratory had been reported negative. The second patient, a man of 45, had symptoms of three months' duration, mimicking duodenal ulcer. At operation a huge ulcerating cancer of the stomach was found.

The gastric chemistry may also be misleading, as shown by a case in a patient of 31 with symptoms of seven months' duration but no abnormal chemical findings. Roentgen examination showed a slightly deformed duodenal cap and a serrated defect on the greater curvature. The case was diagnosed as duodenal ulcer and a Sippy diet was given for a year. Roentgen examination at the end of that time revealed the same defect, and laparotomy advanced cancer.

In another case, in a neurotic woman of 52, with a long history of gastric disturbances and a positive test for occult blood in the gastric contents, an unusually small defect on the lesser curvature was ignored for more than a year, during which the patient was treated for gastritis. At operation a far advanced carcinoma was found.

That misleading roentgen findings may obscure the transitional phase of a benign gastric ulcer into a malignant neoplasm is shown by the case of a man aged 55 who had a gastric ulcer for twenty years. Repeated roentgen examinations showed the ulcer to have become smaller. The patient died from hæmorrhage, and autopsy showed a malignant ulcer with metastasis. In discussing this case the author quotes Alvarez as stating that, "the only way in which one can hope to cure cancer of the stomach is to exercise it during the stage in which it looks and behaves like a benign ulcer". A lesion of the stomach which is apparently healing, as shown by roentgen examinations, may actually be malignant and infiltration of tissue at the base of the defect may occur while the cancer is spreading and give the impression that the niche is filling up.

Causation of Rheumatic Fever

Ryle (*J. A. M. A.*, **131**:1238, 1946) in an address to the Section of Prevention Medicine at the congress of the Royal Sanitary Institute surveyed recent progress in the knowledge of rheumatic fever. He said that evidence is steadily accumulating that Streptococus pyogenes (or certain of its serologic

(*Concluded on page 131*)

NOTES AND NEWS

GUJARAT & KATHIAWAR PROVINCIAL MEDICAL CONFERENCE

The First Gujarat & Kathiawar Provincial Medical Conference under the auspices of the Gujarat & Kathiawar Provincial Branch of the Indian Medical Association will be held during the last week of March 1947, at Ahmedabad. Members from branches of Gujarat and Kathiawar are earnestly requested to participate in the conference and make it a success.

INTERNATIONAL CANCER RESEARCH CONGRESS

The Fourth International Cancer Research Congress will be held in St. Louis, Missouri, U. S. A., during September 2 to 7, 1947. The Union Internationale Contre le Cancer having accepted the invitation of the American Association for Cancer Research, the Congress will be held under the joint auspices of these two organizations, with Dr. E. V. Cowdry, Professor of Anatomy, Washington University School of Medicine and Director of Research of the Barnard Free Skin and Cancer Hospital, serving as President of the Congress.

Of the three Congresses that have been held previously, the first was in Madrid, Spain, in 1933; the second in Brussels, Belgium, in 1936; the third in Atlantic City, New Jersey, U. S. A., in 1939.

In addition to the President, Dr. E. V. Cowdry, Dr. J. Godard, President of the Union Internationale Contre le Cancer and Dr. W. U. Gardner, President of the American Association of Cancer Research, will serve *ex-officio* as members of the Executive Committee.

Headquarters will be at the Hotel Jefferson, St. Louis, where some three hundred rooms will be available for guests. In addition to these rooms, other nearby St. Louis hotels have signified a willingness to make reservations on advance notification by those contemplating attendance at the Congress.

SOYA BEAN

The results of the co-ordinated investigations carried out in the four laboratories at Bombay, Coonoor, Dacca and Lahore, under the auspices of the I.R.F.A., have recently been published as a Special Report (I.R.F.A. No. 13).

The biological value and digestibility coefficient of the soya bean protein by growth and metabolic studies on albino rats and in human beings were found to be of the same order as those of the other pulses. In spite of its high available protein content (1½ to 2 times that of pulses) soya bean did not prove any better than the common Indian pulses as a supplement to the poor rice diet. In most experiments Bengal gram seems to have given bettef results than even soya bean. Incidentally it has been found that the so-called 'poor Madrasi' diet suffers more from a lack of minerals and vitamins than from that of proteins.

One of the methods of processing soya bean for human consumption is to prepare a milk-like fluid from it. Soya bean is softened by steeping for varying periods in warm water and is then ground with further addition of water. When coarse particles are removed by sieving an emulsified fluid is left behind. The emulsion thus obtained looks like milk and in composition varies according to (*a*) the method of preparation and (*b*) the quantity of water added. For ages 'soya milk' thus prepared has been used in China; the main reason for its extensive use in certain regions in China is the almost complete absence of cow's milk from the prevalent human dietary. The soya emulsion prepared as above is inferior even after fortification with salts and vitamins in nutritive value as compared with fresh whole milk. There could be no doubt that an emulsion from the germinated bean would give a preparation with a greater nutritive value as compared with that obtained from untreated beans. There is little proof, however, to show that it approaches cow's milk, although it may appear to be so by virtue of its chemical composition; this is a crude way of comparing two biologicals. The I.R.F.A. Report makes it clear that all the available evidence points to the conclusion 'that soya bean preparations, if fed to infants, need to be supplemented by minerals and vitamins and that even after such supplementation they are less satisfactory than breast milk or cow's milk.—*Indian M. Gaz.*, **81**:444, 1946.

THE NUTRITIVE VALUE OF VARIOUS FATS

Conflicting views continue to be expressed about the nutritional merits of various fats. On the one hand it is suggested that milk fat possesses a specific nutritive quality not present in vegetable fats, while on the other hand some, notably a southern California group, feel that experimental evidence shows that the vegetable oils studied possess all the nutritive qualities of milk fat (butter). Experimental evidence is now presented which seems to show that rats grow equally well irrespective of whether the fat used in the experimental diet is a butter, margarine, corn, cotton seed, peanut or soya bean oil. The evidence appears in a series of observations by Deuel and his co-workers (*J. Nutrition*, **31**:737 & 747, 1946). From the point of view of clinical significance this recent evidence suggests that nutrition may be well served by both milk fat and vegetable fats. From the point of view of economy and general effectiveness milk fat is best taken in the form of good clean whole milk, for whole milk supplies all the valuable nutrient essentials of milk, including butter fat.—*J. A. M. A.*, **131**:1502, 1946.

MEMORANDUM ON B. C. G.: A BRITISH REVIEW

In 1943 the Tuberculosis Association, in conjunction with the Joint Tuberculosis Council and the National Association for the Prevention of Tuberculosis, decided to submit to the Minister of Health a joint request that B. C. G. (Bacille Calmett-Guerin) vaccine should be made available for trial in this country. The following is the general statement from a summary of the memorandum, prepared on behalf of these bodies by Prof. W. H. Tytler, which has now been presented to the Minister of Health and the Secretary of State for Scotland:—

The bodies sponsoring the memorandum ask that the use of B. C. G. should be encouraged, but feel strongly that its employment should not be allowed to discourage the search for more efficient means.

When Calmette advocated the administration of B. C. G. to newborn infants, he believed that it would provide sufficient protection to prevent a natural infection during the first year or two of life, and that sufficient resistance would remain to enable later infections to be overcome and to leave in their turn the higher grade of immunity associated with such recovery. This sequence represents perhaps the most effective result which any form of artificial immunisation can produce. B. C. G. vaccination was advocated especially for

children exposed to special risks, and it has been among these that the most successful results have been claimed in France.

Another application of preventive inoculation is the immunisation of adolescents and young adults who are non-reactors to tuberculin. The value of pursuing this aspect has been shown in the vaccination of young nurses in Norway, where immunisation of non-reactors has been followed by a significant decrease in morbidity and mortality-rates as compared with unimmunised non-reactors, though both rates remain higher in the vaccinated than among the naturally infected reactors. The need for vaccination of this group in Great Britain is shown by the reports of the Prophit Trust investigations. The suggestion is that it should be available, though not compulsory, for non-reactor nurses. This would provide not only a practical safeguard against tuberculosis but a controlled investigation of the first value; there is little doubt that the results would be substantially the same as those obtained in Norway.

The introduction of the transcutaneous method, with its elimination of local abscesses, is felt to be an advance of the first importance; up to 1944 Birkhaug vaccinated 1500 persons with a spring-actuated puncture instrument: 98 per cent became reactors and abscesses developed in none. There appears now to be no reason why B. C. G. vaccination should cause greater inconvenience, or rouse greater prejudice, than does prophylactic immunisation against diphtheria.

It is believed that:

1. The harmlessness of B. C. G. is established beyond doubt.

2. A very considerable degree of immunising efficiency is indicated by the most reliable results from other countries.

3. There exists in tuberculosis services and among the medical profession generally a desire that a reliable supply of B. C. G. vaccine should be available here, as it has been for years in other countries of the world.

4. The application by multiple puncture or by scarification promises to remove the risk of local abscesses, which has been one of the most serious obstacles to the general use of B. C. G.

On these grounds it is requested that the supply and use of B. C. G. vaccine in Great Britain should be facilitated and encouraged.

In the production of B. C. G. for general use it appears to be essential that: (1) the material should be actually safe, (2) there should be public confidence in its safety; and (3) control of production should be such that any claim of tuberculous disease being cue to vaccination should be refutable with certainty. It is believed that, to attain these objects, there should be a single source of supply for Great Britain and that commercial competitions should be avoided. It is also felt that the product should be supported with official backing, by being prepared under the auspices of a Government department of the Medical Research Council, or by certification of the methods employed which should be provided by an official department, such as the standards department of the Medical Research Council. Failing manufacture under Government auspices, it is felt that production should be controlled by some scientific organisation of national repute, which should be subjected to official supervision and certification. Considering that in Norway, during enemy occupation, the number of vaccinations was increased from 603 in 1939 to 17,579 in 1944, it does not seem impossible that an adequate and reliable supply should be forthcoming in Great Britain.—Lancet, **2**:138, 1946.

DANGER FROM ICE-CREAM

Ice-cream is like milk in that it is a perfect bacteriological medium; but, unlike milk, it does not usually start off with a legacy of pathogenic organisms. Of the substances used in its manufacture, milk, dried eggs, and possibly gelatin might contain pathogenic bacteria. Regulations laid down by the Minister of Health should do much to prevent the salmonella in dried egg from causing trouble, and as some form of heat is used in the preparation of most commercial ice-cream the danger from indigenous pathogens is further reduced. In the so-called cold-mix method there are dangers, though paradoxically they may be less than in the hot-mixes, where initial heating often destroys the bacteria present in the original mixture. The chief danger in all types of ice-cream manufacture lies in human contamination, either from dirty hands or from infected droplets coughed into the mixture before it is frozen. In a recent investigation of three outbreaks of staphylococcal food-poisoning caused by ice-cream G. S. Wilson and his colleagues, at Oxford, showed that in all cases the contaminated food had been allowed to stand unfrozen for anything up to 30 hours after preparation. It is during this interval between preparation and freezing that pathogens introduced into a hot-mix find not only a perfect medium for growth but almost equally perfect temperature conditions for rapid multiplication. For this reason the hot-mix method is probably the most dangerous, and the Oxford workers recommended that after preparation ice-cream mix should be frozen almost immediately if pathogens or toxin-producing organisms are to be prevented from multiplying while the mix is slowly cooling down. The remedy, therefore, is some method of immediate cooling, and we are glad to hear that the Ministry of Health is considering compulsory pasteurisation for all types of ice-cream except that made by the cold-mix method. With such regulations in force there is little doubt that large outbreaks like that at Aberystwyth could be avoided.

There remains the problem of getting some workable and reliable method for the bacteriological control of ice-cream. A total bacterial count at 37°C gives some idea of bacteriological cleanliness; but the difficulties of this test may hinder its adoption, and there is need for further study. Many local authorities are also worried because a variety of watery mixtures, sold to the public as ice-cream, may be made in insanitary kitchens, and they seem to have little power to put matters right. The outbreak at Aberystwyth may serve a useful purpose if it forces public opinion to see that such things do not happen again.—Lancet, **2**:277, 1946.

HAZARDS OF THE MEDICAL USES OF RADIATION

DUNLAP (*Occupational Med.*, **1**:137, 1946) in a comprehensive review of the medicolegal aspects of the damage that may result from exposures to roentgen rays, reaium and other radioactive substances describes the hazards under different conditions. Radiation may damage tissue by direct injury to cells and by impairment of the local circulation. For unknown reasons the changes in the tissues become evident some time—weeks or months—after exposure, taking a chronic, slowly progressive course with decrease in healing

(*Concluded at foot of col. 2, page 146*)

CORRESPONDENCE

UNAUTHORISED PRACTITIONERS OF ALLOPATHY

Sir,—I have gone through the article "Unauthorised practitioners of allopathy" in your Journal, May 1946.

May I draw your attention to the fact that unauthorised practitioners of allopathy are not only practising the allopathic system of medicine, some of them have been also appointed and are working as the medical officers of charitable dispensaries getting aids from Government, District Boards, Union Boards and other local bodies. By unauthorised practitioners I mean unregistered doctors of allopathy. According to Bengal Medical Act, sec. 31 unregistered persons are not to hold certain appointment. The sec. 31 is quoted below:—

"Except with the special sanction of the local government, no person other than registered practitioner shall be competent to hold any appointment as Medical Officer of Health or as a physician, surgeon or other medical officer in any hospital, asylum, infirmary, dispensary or lying-in hospital which is supported partially or entirely by public or local funds". Sec. 30 of the act supplies that even if a doctor is duly qualified he must have his name registered before getting a post of any medical officer. It is regrettable to note that there are many dispensaries (in Dinajpur District) having unregistered practitioners as their medical officers. The law is being highly honoured by its breach by the authorities concerned. Should not the Government consider it high time to launch a campaign for replacing the unregistered medical officers by registered ones in charitable dispensaries supported partially or entirely by public and local funds.

I humbly suggest that the State Medical Faculties of all provinces should gather the list of all charitable dispensaries, hospitals and the names of the medical officers with their registration number. Steps should then be taken to replace the unregistered medical officers by registered ones.

I appeal to the members of the State Medical Faculties of all the provinces to take necessary steps for removal of the unregistered doctors from the posts of medical officers of charitable dispensaries. I request them to safeguard the interests of registered doctors for whom and by whom they have been elected as members of the State Medical Faculties. The authorities concerned are requested to do the needful. I am, etc.

Harirampur (Dinajpur)
17-8-46.

Haripada Saha, L.M.F.,
Member, I.M.A., Dinajpur Branch.

SNAKE-BITE AND ANTI-SNAKE-VENOM SERUM

Sir,—From a perusal of the article on "Snake-bite" by Dr. Fazlekarim Khan in the June and July issues of the Journal, it seems that the author and perhaps a great many medical practitioners are not aware of the manufacture by the Haffkine Institute, Bombay, of the "Lyophilised Polyvalent Anti-snake-venom serum" for the past year or so.

It is not only effective against the poison of all the known four varieties of poisonous land-snakes, but it also keeps well for at least 10 years (according to the Haffkine Institute) and it is not essential to keep it in a refrigerator. It costs a little more than the "Anti-venene" manufactured by the Central Research Institute, Kasauli but it well deserves a place in every dispensary.

The medical men and the thousands of our countrymen exposed to the dangers of snake-bite, owe a great debt to Lt. Col. Sir S. S. Sokhey and his batch of research workers for this new improved serum. I am, etc.,

Harigaon (Ahmednagar)
26-8-46.

U. G. Masurkar, M.B.B.S.

WAS PENICILLIN (?) UNKNOWN IN INDIA?

Sir,—I beg to refer to Dr. N. N. Ray's letter in your July issue, appearing under the caption "Was Penicillin (?) unknown in India"? He has very rightly said that a good number of patients suffering from diverse diseases were *said* to have been benefited by the nostrum. Such a 'bread' had come all the way to Rangoon, and as far as I know not one person derived any benefit from it although of course according to rumours many were *said* to have been benefited. There would, of course, be no harm in carrying on a scientific study of the world. I am, etc.

Dhoraji, Kathiawar,
29-8-46.

H. H. V. Lodhia, M.B.B.S.
(Rangoon) D.T.M.

APPRECIATION FOR FREE SUPPLY OF CIBAZOL

Sir,—I have to thank you for publishing our article on "Comparative effects of sulphathiazole and sulphanilamide on small-pox" in Volume XV, No. 10 of the Journal of the Indian Medical Association, July 1946. I may, however, bring to your notice the omission of printing the foot note from the typed copy of our manuscript on the subject sent to you in which we had expressed our grateful appreciation of free supply of Cibazol by Messrs. Ciba (India) Ltd.

I may mention that it was through generous offer of this product (Cibazol) in sufficient quantity without any condition by Messrs. Ciba (India) Ltd. when sulphathiazole could be obtained with great difficulty—that the investigations into the subject could be carried out during the short period continuously without any hindrance.

I have, therefore, to request you to please publish in brief in "Correspondence section" or in any other suitable place in the next issue of the Journal of the Indian Medical Association our grateful appreciation of the generous supply of "Cibazol" by Messrs. Ciba (India) Ltd. free of charge which enabled us to investigate the subject regarding the relative merits of the sulphonamides in the treatment of small-pox.

Thanking you in anticipation of doing the needful in the matter. I am, etc.

City Fever Hospitals,
Arthur Road, Bombay,
3-9-1946.

S. G. Vengsarkar, M.D., F.C.P.S.

J. INDIAN MEDICAL ASSOCIATION

Sir,—May we not demand weekly issues of our Journal of the I. M. A. like those members of the British Medical Association and American Medical Association? It is high time that the Journal should become weekly one and should have informations, all medical as are to be found in the Journals of the British Medical Association and the American Medical Association.

Hope, the matter is taken up at the next All-India Medical Conference and given due consideration in view of the fact that the membership has increased and the paper control (economy) order has also been relaxed. I am, etc.

Patna Medical College Hospital,
Skin & V. D. Dept.,
Patna, 20-9-46.

K. D. Lahiri, M.B.B.S.

M.B.B.S. FOR CALCUTTA M.B.'S

Sir,—I shall be glad to have your views on the undermentioned point which is agitating my mind for the last so many years. This is regarding M.B. degree conferred on the medical graduates of the Calcutta University. It is abundantly clear that most of the universities confer M.B.B.S., degree throughout India, like Bombay, Madras, Lucknow, Lahore, Patna, Hyderabad, etc., and moreover even all the foreign universities confer M.B.B.S., like Hongkong, Singapore, and London while Edinburgh St. Andrews, and Glasgow universities confer M.B.Ch.B., on their medical graduates after completion of their course.

It is correct to say that these alphabets mean nothing but to a private practitioner it has great meaning as it attracts more practice by adding more words from A to Z at the end of one's name.

Now, when Indian Medical Council is trying to have a uniform standard throughout India then why this individual distinction should continue in conferring M.B. degree by Calcutta University.

I would also like to know as to what is the difference, if any, between M.B. (Calcutta) and M.B.B.S., (Madras), or M.B.B.S. (London).

In case there is no difference in training and course then it will be much advisable to confer M.B.B.S., degree on the medical graduates of the Calcutta University as well and this will not only result in bringing uniformity throughout India but the private practitioners as well as the public will be more benefited in one way or the other. I am, etc.

Simla,
25-9-46. P. R. Das Gupta, D.T.M. (Cal.)

MEDICINES OF INDIAN MANUFACTURE

Sir,—It has been suggested and very rightly too that, the Indian medical profession should patronise goods of Indian manufacturers. It has also been stressed that the latter should produce standardised preparations and keep up the standard while at the same time fixing competitive prices as against foreign products. But what is the position in our country today? Hundreds of mushroom firms have sprung up throughout the country during the last six years of war, and most of them have succeeded in making decent profits. Now that the war is over and foreign markets are trying to dump their goods on our country, what is to be the fate of these indigenous concerns? It would be well nigh impossible for most of them to carry on, as they did during the war time when there was a great dearth of imported medicines. I have often pointed out the example of the five biggest pharmaceutical companies in Germany forming themselves into the huge Havero combine after World War No. I and who built up their business and maintained it throughout the world within a short period. If our manufacturers follow suit they would be able to improve their equipment, institute research laboratories, standardise their products and do every thing that will be conducive to the interest of themselves, the profession and the public. I am, etc.

1, Nagindas Mansion,
Girgaum Road, Bombay 4.
3-10-46 (Dr.) U. B. Narayanrao.

MUSHROOMS ! ! ! OPHTHALMOLOGISTS—WHERE ARE YOU ?

Sir,—I would like to draw your attention to your Correspondence Columns. The heading there in October issue of my letter should be

Mushrooms ! ! ! Ophthalmologists—where are you ?

This is a warning to the real ophthalmologist. Here "Mushroom Ophthalmologist—where are you?" does not carry my meaning. I can see that weeds are growing—where should they be ? So will you do something so that my views are properly expressed and no real ophthalmologists have sore eyes. Thanking you, I am etc.

Kumares Chandra Banerjee, M.B.
30-12-46. *Late Sr. H. P.*
Calcutta, *Carmichael Medical College Hospital.*

(*Concluded from page 144*)

power and in resistance to infection. Dunlap lists the most important specific types of injury from radiation under the heads of dermatitis, induction of cancer, necrosis of bone, gastroenteritis, changes in the blood and blood-forming organs, sterility and injury to the fœtus. Dermatitis is the commonest unfavourable reaction and may follow diagnostic, therapeutic and occupational over-exposure. The carcinogenic effect of radiation is almost wholly limited to the induction of carinoma of the skin. Under certain conditions sarcoma of bones may develop from the absorption of radioactive particles, and the suspicion exists that leukæmia may occur more frequently in radiologists than in other comparable groups of persons. Anæmia, leukopenia, thrombocytopenia and other changes in the blood may develop after heavy irradiation of large parts of the body and in poorly protected workers in radiology. Heavy radiotherapy of abdominal tumors may cause serious chronic injuries of the intestine and less frequently of the stomach. "Sterility is a major occupational hazard of both male and female radiologists but can be avoided by proper observance of protective measures". A child exposed to radiation in the uterus may suffer serious malformations, but there is no evidence that exposure to radiation of either parent before conception can hurt the child. Dunlap points out that, while non-medical uses of radiation may present the hazard of over-exposure, most actual injuries have resulted from the improper use of radiation in medical diagnosis and treatment. "Well trained specialists in radiology seldom burn themselves or their patients, and a high proportion of the serious injuries result from the use of radiologic equipment by physicians, surgeons and dermatologists who have a limited understanding of the dangers involved. Any licensed physician regardless of this training may employ roentgen rays and radium in his practice, a situation which has been compared to a general permission to practice medicine with liability only in case of injury. It seems as though some legal restriction might properly be established to limit the use of roentgen rays and radium to persons familiar with their potentialities for good and for evil". Here comes also the question of the liability of the physician who treats cases of curable cancer with ineffective doses of radiation.—*J. A. M. A.*, **131**:1214, 1946.